

THE PROBLEMS OF ANAEMIA IN GENERAL PRACTICE.

THE CLINICAL FINDINGS AND BLOOD PICTURES

IN 86 PATIENTS,

With Special Reference To

Hypochromic Anaemia in Women

of the

Reproductive Period.

by

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## INTRODUCTION.

### Reasons for undertaking the work.

"Happy is the man that findeth wisdom, and the man that getteth understanding." (1).

I graduated in July 1936, and proceeded shortly afterwards to general practice. In the first chaotic weeks when so many preconceived ideas were shattered, I began to doubt the sanity of the authorities who had declared me fit to cope with the thousand problems which humanity was hourly bringing me, in almost pathetic confidence, for solution. As I began, however, with mechanical precision to apply the clinical methods which my teachers had inculcated in me, order began to reign in the chaos. I became increasingly conscious of the fact, that, in the particular practice in which I was engaged, an anaemia of varying degree was probably the commonest symptom complex with which I had to deal.

The patient would present a group of symptoms and signs which could not be explained away except by a diagnosis of 'anaemia.'

Whereas a diagnosis arrived at by the exclusion of every other possible condition is a perfectly legitimate one, especially if it is justified by a satisfactory response to the specific therapy exhibited, this method is open to many objections in the case of the anaemias. A mechanical, instrumental method, with a reasonably small source of personal error, giving the haemoglobin percentage and, preferably, also/

also the blood picture as a whole, is essential, for many reasons. The chief of these are the following.

- (1). The nature of the symptoms and signs presented by the patient who suffers from a mild degree of anaemia, are so vague and indefinite that a blood examination is often the only, and final, proof of the diagnosis.
- (2). The response to treatment in such a case, where no blood count has been done, does not confirm the diagnosis. The obvious fallacy of the extraordinary psychological effect of any form of therapy in certain individuals cannot be ignored.
- (3). Again, in such a mild case the findings of a normal or fairly normal blood picture, at once directs the attention of the physician to some other source of disease. The doctor who diagnoses a mild degree of anaemia (without having recourse to an examination of the blood) to explain the vague clinical findings which may herald the insidious onset of, say, tuberculosis or carcinoma, is accepting a grave responsibility.
- (4). The converse of this statement also holds true - that a blood picture consistently below par, despite adequate therapy, should direct the physician's attention to a more sinister pathology, in a patient whom he has been/

One been treating for a mild degree of anaemia.  
 (5). It is often impossible to classify the type  
 of anaemia without recourse to a blood  
 examination. It is an axiom to state that  
 the first step in the treatment of any case  
 is the establishment of a correct diagnosis.  
 The insistence on a blood examination is the  
 logical conclusion. This is especially  
 obvious in the case of pernicious anaemia.

(6). In estimating the response to treatment the  
 blood examination is usually the only  
 satisfactory method of approach. While the  
 disappearance of oedema, dyspnoea, tachy-  
 cardia, and extreme pallor is obvious in  
 a severe case, in the milder cases the  
 physical signs being less definite, one has  
 often solely to rely on the patient's state-  
 ments. Again the psychological factor  
 cannot be ignored in this assessment.

(7). The instrumental method of gauging the con-  
 dition of a patient's blood is particularly  
 necessary in the follow up of a long-  
 standing case of anaemia (and especially in  
 pernicious anaemia) where a low haemoglobin  
 level - especially if brought about over a  
 period of years - may produce no very urgent  
 symptoms in the patient. (2).

One/  
 the methods used were, for all practicable  
 purposes/



One could augment this list much more fully, but the above were the chief reasons for adoption of what is called a 'side-room technique' in the investigation of certain cases in general practice. They are absolutely obvious, and elementary to any physician. I merely state them because I hope to demonstrate their proof in the cases quoted below, and also because it is an accepted fact as H. Brown ( 3 ) points out, that the average doctor does not do blood counts in general practice. The reason for this is the obviously time-consuming nature of even the simplest method. A single case, according to Brown's estimation (which I think is a modest one) takes an hour to complete.

In view of the surprising frequency with which the anaemia complex was presented to me in the consulting room, I decided that routine blood examination of these cases, would, by the valuable information which it revealed, amply repay the trouble it would entail in a busy working-class practice.

The methods employed are those with which I was acquainted as a student, namely the Sahli Haemoglobinometer and Thoma Haematocytometer. In some cases films were stained and examined by the usual methods and in most cases a diffraction method was adopted, (Allenbury's Halometer) in an attempt to gauge the size of the red blood corpuscles. Eighty-Six cases were examined and it is proposed to show that:-

- (1). The methods used were, for all practicable purposes/

purposes, reasonably accurate, and

(2). in a brief review of the clinical and blood pictures noted in detail below, the findings correspond reasonably well with those of Davidson and the Aberdeen school, whose many papers on the findings in 3.000 individuals will be quoted in detail later.

Description of method of case taking.

TYPE OF PRACTICE IN WHICH WORK WAS DONE.

Selection of Material.

Description of method of case taking.

The practice in which the work was done was situated in a small Fife coastal town of 5.000 inhabitants. It was run by my principal and myself. I went there in October, 1936, and, apart from six months from April till September 1937 (when I held an appointment as a House Physician) I have been continuously employed in the routine duties of an assistant. As neither my deputy in those six months, nor my principal, did any blood counts, the observations given below were all personally made. I owe a great debt of gratitude to my principal for providing instruments, allowing me time off duty, and affording facilities which are not usually accorded an assistant in general practice.

The average patient belonged to what one usually calls the poor classes. Unemployment was rife in the district. Most of the men who had employment were labourers in the nearby quarries and dockyards. Most of the unmarried women, and a few of the married ones, were engaged in the local paper mill. The average wage of the labourers - allowing for spells of unemployment - can be generously estimated at 35/- to 45/- per week. The factory girls, starting at 9/- may rise to a maximum of 33/- per week.

The majority of the patients were either contributors to the National Health Insurance Scheme, or were members of a medical club paying 6d per week.

In/

In selecting the cases for blood examination, the general principle was to collect as many cases of anaemia as possible. No attempt was made to concentrate on one sex, or age group. No discrimination of social status was made. While in most cases the clinical picture suggested a background of anaemia, in some there was no such picture; there was one symptom or sign which might signify an underlying anaemia (e.g. pallor of the face, and nothing else of note). In a still fewer number of cases the patient was apparently normal, and the blood was examined as a control (e.g. against that of another member of the family). The particular indication for blood examination is made sufficiently apparent in each case.

It is proposed at this point to give the clinical notes made on each case. It may be pointed out in objection that the language used is much too colloquial to be employed in a M.D. thesis, but this type of case-taking is adopted with a definite end in view. The scheme adopted is as follows:-

The cases are arranged, for convenience of later reference, in order of age incidence. No opinion, diagnosis, or other comment is offered, unless where it seems particularly indicated, as the cases are dealt with in groups later. Each case history gives the patient's age, sex, and social circumstances. The family menu is indicated and in some cases, described/



described in detail. The patient's history and complaints - as far as possible reported in his own words - are then given. The physical findings are then noted. Only positive findings are recorded except in a few cases where a negative finding was particularly striking. If there is still no indication for a blood examination this is now stated. Then follow the blood examination, the treatment prescribed, and the progress notes, with further blood counts, if any have been done.

Case 1. Agnes 2. set 3.

This was a pale little girl, whose mother brought her, saying that she had been off colour lately. I had been attending her little brother for an attack of acute bronchitis, and it was suggested when I called that I should also examine her.

CASE NOTES OF 86 INDIVIDUALS EXAMINED,

21st October 1936 arranged in order of age. The following:-

Hæmoglobin.	79.
R.B.C.	4,490,000.
G.I.	0.89.
Salometer reading.	7.2 u.

I prescribed Ferri. et. amm. cit. gr. x, t.i.d., and suggested that she take an orange at least every second day. She appeared to tolerate this dose of scale iron very well, and her mother told me, when she saw me two weeks later, that Agnes was very much improved and taking her food much better. The iron therapy was continued for eight weeks and her blood picture on 7th December, 1936, was as follows:-

Hæmoglobin.	94.
R.B.C.	5,040,000.
G.I.	0.94.
Salometer reading.	7.3 u.

I saw her two months ago, when she appeared to be in perfect health.

Case 2. Ene V. set 3.

This little girl was brought because she had colicky pains in the abdomen, was off colour, and would not/

Case 1. Agnes S. aet 3.

This was a pale little girl, whose mother brought her, saying that she had been off colour lately. I had been attending her little brother for an attack of acute bronchitis, and it was suggested when I called that I should give Agnes a tonic. A blood count was done on 21st October, 1936, which revealed the following:-

Haemoglobin.	79.
R.B.C.	4.490,000.
C.I.	0.89.
Halometer reading.	7.2 u.

I prescribed Ferri. et. amm. cit. gr. x, t.i.d., and suggested that she take an orange at least every second day. She appeared to tolerate this dose of scale iron very well, and her mother told me, when she saw me two weeks later, that Agnes was very much improved and taking her food much better. The iron therapy was continued for eight weeks and her blood picture on 7th December, 1936, was as follows:-

Haemoglobin.	94.
R.B.C.	5,040,000.
C.I.	0.94.
Halometer reading.	7.8 u.

I saw her two months ago, when she appeared to be in perfect health.

Case 2. Ena V. aet. 3.

This little girl was brought because she had colicky pains in the abdomen, was off colour, and would not/

not eat her meals with her usual gusto. She was blonde and had a very fair transparent skin. The cheeks and mucous membranes were pale. She had threadworms in her stools. The examination of her blood gave the following result:-

23.11.36.

Haemoglobin.	81.
R.B.C.	4,730,000.
C.I.	1.07.
W.B.C.	11,060.
Halometer reading.	6.4 u.

The stained film gave a differential count as follows:-

Polymorphs.	49%
Lymphocytes.	31%
Eosinophils.	15%
Mononuclears.	4%
Basophils.	1%

The child was given Butolan tablets, and in a fortnight was back to her usual good health.

### Case 3. Robert G. aet 3.

This little boy had been the subject of repeated attacks of bronchitis for the preceding year. He was just recovering from an attack when examined. He looked pale but the mucous membranes of the mouth and eyes were not blanched. A heel stab was done on December, 1936. He kicked over a whole tableful of apparatus and broke an R.B.C. pipette but the haemoglobinometer was saved from the wreckage and gave a reading of:-

Haemoglobin.	68.
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I prescribed Ferri. et. amn. cit. gr. iii, t.i.d. when he tolerated very well. He took this medicine intermittently (four weeks on, and two weeks off) from December 1936 until October 1937. Child's general health.

He certainly had a very much better winter and had fewer attacks of bronchitis. His mother was very enthusiastic about the results of iron therapy, so much so that she induced him to have another blood examination, with the following results:-

22.10.37.

Haemoglobin.	110.
R.B.C.	5.240,000.
C.I.	1.06.
Halometer reading.	7.1 u.
W.B.C.	13.200.

The differential count was as follows:-

Polymorphs.	70%
Lymphocytes.	25%
Mononuclears.	12%
Eosinophils.	2%
Basophils.	1%

There was no relative or actual lymphocytosis and the chest condition was, in my opinion, purely pyogenic. There was no evidence of tuberculosis and, indeed, apart from the physical signs in the chest, the child was singularly robust and healthy.

#### Case 4. David N. aet 5.

I was called in to see this little boy whose mother gave me this history that he had been "off colour and out of sorts for three weeks". She had noticed/



noticed threadworms in his stools and had given him "worm tablets". These had a good effect, but although she had seen no worms for the past week she had not noticed much improvement in the child's general health.

Apart from the fact that the mucous membranes were pale I could detect no evidence of organic disease. I had not suspected anaemia and merely did the blood count as a control. The examination of the blood on 27th November, 1936 revealed the following:-

Haemoglobin.	58.
R.B.C.	3,530,000.
C.I.	0.83.
Halometer reading.	7.5 u.

The blood film showed nothing of note. The cells appeared to be large and well filled with haemoglobin. A leucocyte count was not done. Further questioning revealed the fact that this child had pale, bulky, and greasy stools. I prescribed Glaxo Ferrous sulphate gr. iiii, t.i.d. and in two weeks he was clinically much improved. The mother was most enthusiastic about the treatment.

I saw this patient fortuitously a year later, and induced him to come for another blood test. His mother brought him up and the following result was obtained:-

10.2.38.

Haemoglobin.	78.
R.B.C.	4,940,000.
C.I.	0.80.
Halometer reading.	7.4 u.

His mother stated that for the past year David had been in perfect health and had not missed one day's school/

school attendance. four weeks, the improvement in his

He is an only child and there is no question of any dietary deficiency.

Case 5. Billy H. aet 5.

This was a pale faced little boy. He was just recovering from an attack of whooping cough, which responded very well to a mixed pertussis vaccine. The paroxysms ceased four days after the first injection, i.e. on the day following the second injection. He developed a subacute bronchitis despite the vaccine, but this was very mild and there was no pneumonic consolidation. His little brother, aged three, was given the vaccine prophylactically, and did not develop any cough.

Billy was very pale and the conjunctivae (after the coryza stage of whooping cough had subsided) were very pale. He had no other symptom or sign suggestive of anaemia and a blood examination on 19th October, 1937 revealed the following:-

Haemoglobin	71.
R.B.C.	4.930.000.
C.I.	0.72
W.B.C.	10.600.

This count was taken six weeks after the onset of whooping cough, while he still had a subacute bronchitis. He was given Ferrous sulphate gr. iiii, t.i.d. and improved in general health.

It was interesting to note that after he had taken/

taken the pills for four weeks, the improvement in his general health coincided with a change in his complexion. While there was still pallor of a marked degree, the skin had a healthy glow and had lost its unnatural "pasty" appearance. This change was noted in many of the cases noted below. The conjunctivae were still pale, and I have, indeed, throughout this series of cases, been unable, except in severe cases, to find any certain correspondence between the pallor of the conjunctivae and mucous membranes, and the degree of anaemia present.

Case 6. Bobby S. aet 6.

Again a pale little boy, rather undersized and under-nourished, who was brought to me because of an infestation with thread worms. These yielded rapidly to Butolan therapy. I examined his blood one week after the first consultation and found the following:-

14.11.37.

Haemoglobin.	69.
R.B.C.	4.280.000.
C.I.	0.81.
W.B.C.	10.900.

The differential count was as follows:-

Polymorphs.	55%
Lymphocytes	20%
Mononuclears	23%
Eosinophils.	2%

There was a large percentage of mononuclear cells but no eosinophilia. He was given Ferrous sulphate gr. iii, t.i.d. and reported three weeks later, looking and feeling very much improved. He failed to report again./

again.

Case 7. Jessie T. aet 6.

Mrs. T. came to consult me about her son Jim (vide infra, case 11), and as Jessie came with her I arranged to examine her blood as a control against her brother. She appeared to be in perfect health and had no complaints. The examination revealed the following:-

24.10.37.

Haemoglobin.	75.
R.B.C.	4.620.000.
C.I.	0.82.
W.B.C.	6.400.

She was advised to take Ferrous sulphate gr. 111, t.i.d. and I did not see her again for some time.

23.2.38.

I saw Jessie one week ago when she had the following symptoms. She had during the past two weeks a marked loss of appetite. She would only take a few mouthfuls and feel "full up". One or two hours later she felt ready for a meal once more. For the past three days the taking of any food precipitated an attack of colicky pain in the abdomen, associated with diarrhoea. On inspection the abdomen was found to be tumid and free fluid was demonstrated. The Morro's cutaneous test was positive. In view of the findings, and also those stated below in reference to her brother, I had her seen by a specialist who diagnosed "tabes mesenterica". She is on the waiting list of a surgical ward of the Royal Infirmary of Edinburgh.

Case/



Case 8. James L. aet 7.

This little boy was brought to see me because of vague complaints of anorexia, constipation and lassitude. He was an only child and his mother was of the over-anxious, worrying type. She said "continually catching colds". He looked very pale, and was, altogether, a miserable little fellow. Apart from two very carious teeth (which I had extracted) I could find no evidence of any organic disease.

I examined his blood and found the following:-

16.10.36.

Haemoglobin	69.
R.B.C.	4.980.000.
C.I.	0.69.
Halometer reading.	7.4 u.

I prescribed Ferri. et. amm. cit. gr. xx, t.i.d. and he tolerated this large dose very well. I saw him one week later. His mother said he was much the better of his tonic. He was eating much better. She was, however, very impressionable, and I did not put very much stress on the alleged improvement. His blood examination revealed the following:-

23.10.36.

Haemoglobin.	69.
R.B.C.	5.480.000.
C.I.	0.64.
Halometer reading.	7.4 u.

I asked her to continue with the tonic and report in four weeks. She returned with James on 22nd November, 1936 (she was one of the very few patients who did exactly as she was bid) and the figures found on that date/



date were as follows:-

22.11.36.

Haemoglobin.	98.
R.B.C.	5.120.000.
C.I.	0.96
Halometer reading.	7.2 u.

James was looking very much more fit. His cheeks had a healthy glow and his appetite, I was informed, was voracious.

There was a definite element of dietary deficiency in this case. The actual intake was sufficient as regards calories, but, although Mrs. L. lavished a great deal of care over her only son, his meals were deficient in first class protein and vitamins because of the higher price of these foodstuffs. He has now left the district and I have no more notes on his case.

Case 9. Margaret R. (aet 7).

This little girl had a sore throat and when I went to see her I found that she had very markedly enlarged tonsils and adenoids. She had a typical adenoid facies. Her mother informed me that she had had repeated attacks of sore throat or bronchitis for the last three winters. Her tonsils were of the large pale variety. There was no glandular involvement, no history of rheumatism or any cardiac mischief. She improved very quickly with gargles and a throat paint. I examined her blood one week after her symptoms had subsided and found the following:-/

following:-

16.10.36.

Haemoglobin	58.
R.B.C.	4.620.000.
C.I.	0.63.
Halometer reading.	6.8 u.

Glaxo Ferrous sulphate gr. 111, t.i.d. was prescribed. I saw her three weeks later when she was very much improved. Her appetite was voracious and her mother was most enthusiastic about the "iron pills." She failed, however, to report and, when I called after one month, Mrs. R. told me that she thought Margaret need not come back or take any more medicine, as she was in perfect health. I induced her to come for a blood examination which revealed the following:-

7.2.37.

Haemoglobin	72.
R.B.C.	4.250.000.
C.I.	0.86.
Halometer reading.	7.3 u.

I advised her to take one three grain ferrous sulphate pill three times a day for one month. In June 1937 Margaret had her tonsils and adenoids removed, and was given one month's course of Ferrous sulphate after this as a tonic.

I did not see her again until October 1937, when the condition of her blood was found to be as follows:-

31.10.37.

Haemoglobin.	91.
R.B.C.	4.920.000.
C.I.	0.93.
W.B.C.	8.600.

In/

In the period of October 1936 - October 1937, Margaret had taken three courses of Ferrous sulphate gr. iiii, t.i.d. for one month. She suffered from astigmatism, and when her mother brought her to me for advice about a developing strabismus, I took the opportunity of examining her blood and found -

5.3.38.

Haemoglobin.

86.

It may not be irrelevant to note at this point that, in the opinion of the present writer, the results of iron therapy in general practice are often less satisfactory than one would expect, because of the difficulty which the practitioner experiences in

- (1) persuading the patient to take an adequate amount of the particular iron preparation over an adequate period of time;
- (2) persuading the patient to report for follow-up blood counts.

The delinquents fall into two distinct classes, viz. the longstanding moderately severe or severe cases which are not showing much improvement; and those cases which have recently shown a satisfactory response. Apart from the cost, palatability, and various gastrointestinal disturbances associated with the exhibition of any particular preparation (these will be referred to in detail later) a certain amount of dulling of the mental faculties from a longstanding minor degree of cerebral anoxaemia is, in my opinion, a frequent source of failure of co-operation between doctor and patients in the first group of patients. The second group simply refuse to carry on with medicine when they feel in perfect health. The admonition that relapses will possibly occur are waived with scorn. The patient goes off, well pleased with the dramatic diminution or disappearance of symptoms, and fails to report again. Such an attitude is not always sheer perversity and base ingratitude, but is, most often (at least in the particular practice in which this work was done) dictated by the patients' financial circumstances. A consultation and blood examination was a luxury of the first order for many of the patients whose income could scarcely allow even the price of a bottle of medicine.

Case 10. Elizabeth P. aet 7.

This little girl is the daughter of one of the better class patients of the practice. There was no question of any dietary deficiency. She was, at the time of examination, confined to bed and convalescing (3rd week) from a mild attack of faucial diphtheria. She received 10.000 units of antitoxin on the second day, the diagnosis was confirmed bacteriologically on the following day. She was not removed to hospital as adequate quarantine and isolation could be maintained at home. The blood examination three weeks after the onset revealed the following:-

24.11.36.

Haemoglobin.	46.
R.B.C.	4.110.000.
C.I.	0.56.
Halometer reading.	7.1 u.

She had a pronounced tachycardia, but she had no evidence of any nervous system involvement.

She was given Glaxo Ferrous sulphate gr. 111, t.i.d. and with rest in bed made a rapid and uneventful recovery, which was sustained. The blood examination was not repeated as it was considered unnecessary.

Case 11. Jim T. aet 9.

This little boy is the brother of Jessie T., case 7. He is one of a family of four. His father and mother are alive and well. Mr. T. is a foreman in the local papermill and there is no question of dietary deficiency/



deficiency. Mrs. T. has very marked scars in the submandibular region, the result of incision for supposedly tuberculous glands in childhood.

Jim was brought to me with a history of anorexia, constipation alternating with diarrhoea, loss of weight, flatulence and occasional vomiting. On examination it was observed that Jim was a pale, thin boy. He had a sallow muddy complexion, His pulse rate was 106 per minute. On inspection it was found that the abdomen was slightly enlarged and seemed unduly prominent for his general build. There was a history of colicky abdominal pain. On palpation I detected a curious "doughy" sensation which is alleged to be characteristic of "tabes mesenterica." Deep pressure elicited pain in both iliac fossae. I could detect no free fluid in the abdomen. The urine showed no abnormality. The examination of his blood revealed the following:-

24.10.37.

Haemoglobin.	58.
R.B.C.	4.370.000.
C.I.	0.67.
Halometer reading.	7.6 u.
W.B.C.	8.200.

The differential count was as follows:-

Polymorphs.	70%
Lymphocytes	26%
L.Mononuclears.	3%
Eosinophils.	0%
Basophils.	1%

He was referred to the Royal Infirmary of Edinburgh. A diagnosis of appendicitis was made. The underlying/



underlying pathology was alleged to be due to tuberculous glands in the ileocaecal area and it was stated that ascites of mild degree had been demonstrated.

The diagnosis was confirmed radiologically.

He was notified as a tuberculous case. Tuberculin ointment was obtained and Morro's test was frankly positive. The other two children were also positive. (The fourth member of the family, Betty aet 6, is in a mental defective home, her condition being due to a cerebral haemorrhage at birth - Jessie (case 7) is her twin.).

Jim was given Ferrous sulphate gr. iiii, t.i.d. which he tolerated well. His general health is much improved and he is attending school regularly. He is still on the waiting list of the Royal Infirmary, for treatment of the abdominal condition which appears to be settling.

#### Case 12. Walter S. aet 9.

This patient came under my notice as the result of an attack of acute gastritis.

He is one of a family of two, who lives with his parents in a single room. The father is unemployed; his mother is a dull, weary woman who has long since had her spirit crushed by the unequal odds in a hard struggle for bare existence. The family menu consists largely of tea, bread, margarine, corned beef, half to one/

one pint of milk daily, potatoes, a cheap bacon "fry" once a week - no fruit, practically no vegetables, eggs only in the summer months. The chief deficiency is in protein, vitamins and mineral salts.

Walter had an acute gastritis with associated diarrhoea, which yielded rapidly to symptomatic treatment. He had six carious teeth, which I had extracted. He rapidly returned to his usual "good health". He was pale, undersized and underweight, and was a nervous, highly-strung child. There was no organic lesion demonstrated. The examination of his blood two weeks after the gastro-enteritis had settled revealed the following:-

15.11.37.

Haemoglobin.	80.
R.B.C.	5.350.000.
C.I.	0.75.
W.B.C.	6.200.
Halometer reading.	6.9 u.

He was given Ferri et. ammon. cit. gr. vii t.i.d. In fourteen days he was clinically much improved. I prescribed a cod liver oil emulsion and saw him one month later. He was a very much happier and more robust little fellow and his appetite was greatly improved.

Case 13. James M. (aet 9).

This was a healthy schoolboy who presented no evidence of disease. He was an only child in a good-class comfortable home, and I arranged to examine his blood/

blood as a control at the same time as I did that of his mother, as I had attended him one month previously for an attack of follicular tonsillitis. The result was as follows:-

5.12.36.

Haemoglobin.	90.
R.B.C.	5.540.000.
C.I.	0.82.
Halometer reading.	6.8 u.

Case 14. Duncan C. aet 9.

This little boy is one of a family of three. The father is a sailor. I have called at meal times and am convinced that a generous mixed diet is the daily menu. Duncan was a pale little boy, rather undersize for his years (his brother aged 7 is  $\frac{1}{2}$ " taller). He was rather puffy under the eyes. The urine had no abnormal constituent. There was a history of scarlet fever and of frequently recurring sore throat. He had rheumatic fever at the age of six. There was no history of frank nephritis. The boy had always been nervous and highly-strung. He was always "on edge" and continually "up to something", his mother told me.

On examination I found, as stated above, that he was very pale and had puffy lower eyelids. The mucous membranes of the eyes, lips, and gums were pale. There was no marked tachycardia (84 per minute) or any evidence of a cardiac lesion.

As I examined the boy I noticed that he was very garrulous/

garrulous. He was rather unstable emotionally, and could be induced to laugh or cry very easily. There was an occasional spasmodic twitching of the corner of the mouth on the left side. He performed finer movements, such as buttoning his jacket, with difficulty and after much fumbling. He was in my opinion a mild case of chorea.

I prescribed aspirin gr. v, t.i.d. for one month and improvement began very quickly - too quickly, in my opinion, to be attributable to salicylates. The aspirin was stopped after four weeks and no further specific therapy (salicylate or otherwise) was exhibited.

He was then given Syrup Minadex 3 i, t.i.d. and his blood examined at the end of one fortnight. The result was as follows:-

28.10.36.

Haemoglobin	60.
R.B.C.	4.750.000.
C.I.	0.64.
Halometer reading.	7.4 u.

Ferrous sulphate gr. iiii, t.i.d. was prescribed, and at the end of one month the blood examination revealed the following:-

26.11.36.

Haemoglobin.	83.
R.B.C.	4.570.000.
C.I.	0.92.
Halometer reading.	7.5 u.

He had two carious teeth which I had removed. He was advised to continue with the iron pills.

I saw him after one month and at repeated intervals. The/



The nervous manifestation had gone entirely after two months. Although the child remained very pale the puffiness of the eyes vanished, the appetite improved and he appeared to be in excellent health.

He was advised to take Ferrous sulphate tablets for one month, then miss two months, then for a further month, and so on. On giving him a complete overhaul one year from my first examination I could only pronounce him to be in perfect health. The blood examination revealed the following condition:-

23.10.37.

Haemoglobin.	85.
R.B.C.	4.900.000.
W.B.C.	7.200. less readily
C.I.	0.85.
Halometer reading.	7.2 u. and other

Case 15. Elizabeth S. aet. 10.

This girl is the daughter of the local mill owners and belongs to one of the most financially favoured families in the district. There was no question of dietary lack.

Elizabeth was brought to my notice with a history of being easily tired. She had a marked loss of appetite and lack of vitality. It was suggested that I give her a tonic.

She looked very pale, the mucous membranes were moderately pale but apart from a mild degree of acne vulgaris there was nothing else of note. The examination of the blood revealed the following:-

28.10.36/



28.10.36.

Haemoglobin.	78.
R.B.C.	4.770.000.
C.I.	0.83.
Halometer reading.	7.5 u.

I gave her a prescription for Syrup Minadex 3,

i. t.i.d. After one month the findings were as follows:-

9.12.36.

Haemoglobin.	70.
R.B.C.	4.780.000.
C.I.	0.74.
Halometer reading.	6.6. u

Her appetite was very much improved and the general opinion of the family was that the tonic had done her a "lot of good". I prescribed Ferrous sulphate gr. iiii, t.i.d. and after one month she was very much improved. She was eating much better, was less readily tired, and keen to participate in hockey and other school games. A further blood examination gave the following results:-

3.2.37.

Haemoglobin.	90.
R.B.C.	4.950.000.
C.I.	0.91.

Throughout the winter of 1937 the child has been in much better health and the mother is most enthusiastic about the iron pills.

Case 16. George W. aet 13.

This was an only child. His mother was of a particularly anxious and worrying temperament. Her blood count is given below (case 42).

George had been attending me two months previously/

previously for a mild attack of bronchitis, which cleared up satisfactorily. Syrup Minadex was prescribed as a tonic and his mother brought him up for an opinion on his general fitness for school.

He looked rather pale and had a stupid, inane expression, the relic of markedly enlarged adenoids in infancy.

Apart from his pallor there was nothing abnormal detected. The blood findings were as follows:-

1.11.36.

Haemoglobin.	99.
R.B.C.	5.470.000.
C.I.	0.92.
Halometer reading.	7.0 u.

Case 17. Douglas G. (aet 13).

This was a miserably thin, pale-faced boy whose mother brought him to see me on account of borborygmi.

As well as the pallor it was noted on examination that he had a very sallow muddy complexion. The mucous membranes were very pale.

He had a history of rheumatic fever and repeated attacks of pharyngitis and tonsillitis. He had a mitral stenosis evidenced by a presystolic murmur in the mitral area. Apart from the general impression of under- or mal-nutrition, I could find nothing else of note. The examination of the abdomen was negative. On personal enquiry I found that his dietary arrangements were far from physiological. He was an only child and his mother, a widow, was out all day acting as a charwoman. His meals/

meals were prepared in a haphazard manner and tea, buns and pastries featured largely in almost every meal. He ate very little fruit, had few decently cooked meals, with at least one fried course daily. The actual income of this family of two, was probably, quite sufficient to maintain a suitably balanced and mixed, generous diet. The domestic arrangements were probably to blame for the ineffective management. The general result was a high fat, high carbohydrate astringent diet (too much tea) with poor class, or twice cooked proteins, low in vitamin and mineral salts, and with too little residue of intestinal roughage to maintain and promote regular peristalsis.

The examination of the blood revealed the following:-

7.11.36.

Haemoglobin.	74.
R.B.C.	4.610.000.
C.I.	0.80.
Halometer reading.	6.8 u.

Douglas was prescribed Ferri. et. ammon. cit. gr. xx, t.i.d. and one orange daily. His mother was advised about his dietary requirements.

He made little progress and six weeks later I referred him to the Royal Infirmary of Edinburgh. There a tentative diagnosis of tabes mesenterica was made. He was admitted as an in-patient and improved greatly with the usual hospital routine. The diagnosis, however, was not established. He was discharged very much/

much improved. I saw him about one year later when he scalded his arm. The blood examination was as follows:-

23.10.37.

Haemoglobin.	68.
R.B.C.	4.540.000.
W.B.C.	6.010.
C.I.	0.76.
Halometer reading.	7.2 u.

Ferrous sulphate gr. iii, t.i.d. was prescribed. He improved rapidly, and approximately four weeks later the reading was:-

18.11.37.

Haemoglobin.	80.
R.B.C.	5.040.000.
C.I.	0.80.
Halometer reading.	7.3 u.

He was looking and feeling much fitter. He had a rosy tinge in his cheeks and was attending school very regularly.

My latest blood examination showed a further slight improvement:-

7.2.38.

Haemoglobin.	86.
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Case 18. Jean McN. aet 16.

This girl is a shop-assistant. She is the only daughter, and youngest of a family of three. Her father is a joiner earning an adequate income, and there was no evidence of dietary deficiency. The menarche occurred at the age of 14. The periods have been regular and apparently normal. The patient had a tuberculous pleurisy in childhood and was regarded by her /



her mother as being "rather delicate". She was, consequently, having her diet continually reinforced with tonics and various proprietary cod liver oil preparations. She was very susceptible to winter cough, and I first saw her during an attack of acute bronchitis. She cleared up satisfactorily in two weeks and although there were a few ronchi in patches scattered over the chest, her pulse was regular and of good volume, and there was no indication of a tuberculous focus in the lungs.

She quickly recovered and returned to work. I saw her two months later. There was no evidence of bronchitis. She was complaining of lassitude, loss of energy and lack of vitality. She had a "pink and white" complexion; the mucous membranes were not unnaturally pale. There was no tachycardia.

Examination of her blood revealed the following:-

24.10.36.

Haemoglobin.	75.
R.B.C.	4.670.000.
C.I.	0.82.
Halometer reading.	6.9 u.

She was given Ferrous sulphate gr. 111, t.i.d. and advised to take one orange daily. She returned in one month's time after taking 100 pills. She was looking and feeling much better. She said she felt more fit for her work, and altogether was in a much happier frame of mind. The blood figures were as follows:-

29.11.36./

Haemoglobin.	75.
R.B.C.	4.670.000.
C.I.	0.82.
Halometer reading.	6.9 u.



29.11.36.

Haemoglobin.	96.
R.B.C.	5.070.000.
C.I.	0.96.
Halometer reading.	7.0 u.

She said she found the pills quite easily taken and had suffered no digestive upset.

I saw her one year later when she came complaining of pain in her foot (tendo calcaneus bursitis). She appeared to be in perfect health apart from this, and was asked to report for a blood count but failed to do so.

Case 19. Jim H. aet 16.

This boy is a painter's apprentice. He reported to me with a story of vague abdominal pains. There was no other complaint. There was no history of constipation. On examination it was noticed that he looked pale, but he had always done so. The mucous membranes were pale. The teeth were healthy and there was no sign of any blue line of plumbism on the gums. There was no evidence of any weakness of the extensor tendons of the wrist or any abnormality in the central nervous system.

In view of his occupation, however, and the history of colicky pain, it was decided to examine his blood. The following result was noted.

14.11.37.

Haemoglobin.	103.
R.B.C.	5.240.000.
C.I.	0.99.
W.B.C.	7.400.
Halometer reading.	6.9 u.

The/

The stained film showed no punctate basophilia or any other abnormality.

The patient was given Tincture of Belladonna M vii, t.i.d. with a very good result. He was entirely relieved in three days and the belladonna was stopped. He reported at weekly intervals for one month but no further symptoms developed.

Case 20. Annie L. aet 19.

This girl worked as a clerkess in one of the local shops. Menarche occurred at the age of 13. Her periods were irregular and scanty but she had no dysmenorrhoea, or leucorrhoea. She came to see me at her mother's behest, to get a tonic. She said she felt quite well except that at the end of the day she felt rather tired. She was very pale and the mucous membranes were pale. There was no tachycardia. There was nothing of note to be made out by clinical examination. The blood examination gave the following result:-

29.12.36.

Haemoglobin.	77.
R.B.C.	5.030.000.
W.B.C.	7.400.
C.I.	0.77.
Halometer reading.	6.9 u.

She was given a prescription for :-

Ferri. et ammon. cit. gr. xv. )	
Tr. nucis. vom. M v. )	t.i.d.

She reported two weeks later to have her bottle refilled/

refilled and said she felt much better. She had no digestive upset, but complained rather bitterly about the staining effect of the iron on her teeth.

She reported in a further two weeks, looking and feeling very much improved. She stated that she was slightly constipated. She felt much better and more fit for her work.

She failed to report again.

Her father is a local contractor, earning a comfortable income. The mother's case is quoted below (case 72). Annie is one of a family of two and I do not think there is any question of dietary deficiency, although the actual intake may have been below standard on account of anorexia.

Case 21. Georgina R. aet 19.

This girl was a factory hand in the local paper mill. She lives with her father and mother in a comfortable three-roomed house. Her two brothers are grown-up and married. The mother's blood examination is quoted below (case 73). I have called at meal-times and, in my opinion, this patient has the choice of a fairly well-balanced diet.

Menarche - aet 13. The periods were always regular and apparently normal, no dysmenorrhoea. This girl is the youngest of the family and very "spoiled". She has many and various food fads, tastes and dislikes. Her/

Her mother, who is herself of neurotic temperament, is apt to give in to her in many things. It is a frequent occurrence, for example, for Georgina to be off work and in bed for the first two days of her menstrual period, although I am convinced from personal observation, that there is no dysmenorrhoea. When I examined her she complained of lack of appetite, sleeplessness, loss of energy, and inability to concentrate. She had attended one year ago with severe chilblains but there was, at this time, no evidence of them. She was pale and the mucous membranes were blanched. She had a pulse of 98 per minute, but there was no evidence of any cardiac lesion.

The examination of her blood revealed the following:-

1.11.37.

Haemoglobin.	64.
R.B.C.	3.700.000.
C.I.	0.87.
Halometer reading.	7.2 u.

Ferri. et ammon. cit. gr. xx, t.i.d. was prescribed.

This caused marked constipation but was relieved by symptomatic treatment, and the iron continued. She did not feel very much better when I saw her two weeks later.

The blood examination three weeks after the commencement of treatment revealed the following:-

24.11.37.

Haemoglobin.	82.
R.B.C.	4.590.000.
C.I.	0.86.
W.B.C.	7.200.
Halometer reading.	7.3 u.



I found out on this occasion that she had been smoking 10-15 cigarette per day. She had been worried lest this was the cause of the "bloodlessness" as her mother alleged it was. The furtive partaking of the forbidden fruit had been a source of conflict and general upset in the nervous system of this highly-strung adolescent girl. I had a talk with her and she seemed more assured.

The improvement of her blood figures was rapidly followed by an improvement in her general condition. Most noticeable of all was the improvement of appetite. She was induced to take a generous mixed diet, and continued on scale iron for two months.

I saw her at the end of two months. Her cheeks were rosy; she looked healthy and happy, but she unfortunately failed to report for a further blood examination.

Case 22. Elizabeth S. aet 20.

This girl is a shop assistant. Menarche occurred at the age of  $13\frac{1}{2}$  years. Her periods are fairly regular, lasting 3 - 4 days and there is no dysmenorrhoea. She came complaining of a sore throat. On examination a simple follicular tonsillitis was demonstrated. There was a good deal of faucial congestion and two carious teeth were observed. She gave a history of recurrent attacks of sore throat, but there was no other evidence of any other possible rheumatic manifestation. There was no cardiac lesion.

She/



She had a tachycardia 95 - 110 which persisted after the naso-pharyngeal condition had settled. She gargled for the first ten days with Liquor Alkalini B.P. and then the following was given:-

Liq. Ferri. Perchlor.	M xx.
Liq. hydrarg. perchlor.	M c.
Pot. chlor.	gr. v.
Liq. strych. hydrochlor.	M v.
Aq. ad.	zp.
3 ii ex aqua Q.I.D. gargle and swallow.	

She was thus getting Liq. Ferri. perchlor. M x, Q.I.D.

After one week on this gargle the local symptoms were markedly relieved but she looked very pale and was feeling out of sorts generally. The mouth and throat were perfectly normal (the carious teeth were extracted) and the only positive physical signs were pallor of the mucous membranes and tachycardia. The examination of the blood revealed the following:-

19.10.37.

Haemoglobin.	72.
R.B.C.	4.560.000.
C.I.	0.80.
W.B.C.	9.400.
Halometer reading.	7.0 u.

She was at that time at work as a shop assistant. Her appetite was slightly impaired, but she was taking a diet comparatively rich in iron and had also been taking orange juice daily. I prescribed Liq. Ferri. Perchlor. M x ex aqua, t.i.d. She tolerated this very well. She did not seem to mind the taste and there was no upset of the alimentary system.

I/

I saw her and examined her blood exactly one month later, to find the following:-

19.11.37.

Haemoglobin.	70.
R.B.C.	4.480.000.
C.I.	0.79.
Halometer reading.	6.8 u.

I then prescribed Ferri. et. ammon. cit. gr. xxx, t.i.d. but she could not tolerate this dose. It occasioned the onset of acute diarrhoea. I halved the dose and gave her gr. xv, Q.I.D. (gr. 60 daily). She tolerated this quite well and continued taking it for 6 weeks. I examined her two months after the previous blood count. She was looking much more fit. The mucous membranes were not so pale. Her cheeks had a healthy glow and she herself said she felt much more fit for work. She said she had realised how "run down" she must have been previously. The most striking improvement she thought, was in her appetite, which was now voracious. The blood examination revealed the following:-

30.1.38.

Haemoglobin.	94.
R.B.C.	5.040.000.
C.I.	0.94.

Case 23. Margaret C. aet 20.

This girl is the youngest of a family of four. She is employed in the local paper mill. She belongs to a very thrifty, thriving family, who have a nice three-/-

three-roomed house in one of the better class areas in the town. Margaret, who is a chum of Georgina (case 21) is also the youngest member of the family, has an anxious, worrying mother, and is subject to an almost identical environment. She does not, however, display the same emotional instability, and is most anxious to get back to work.

The menarche occurred at the age of 15 years. The menstrual periods have always been irregular and menstruation itself scanty. There is no history of dysmenorrhoea or leucorrhoea. Amenorrhoea was marked and was in point of fact her leading symptom. Unfortunately, Margaret had been on Ferri. et. ammon. cit. gr. xv, t.i.d. for three months before she came to my notice.

When I examined her she had had one period in the previous three months and her next period was three weeks overdue. She looked pale. Her mucous membranes were very pale. She was of sallow complexion and she had an oily skin with a mild degree of seborrhoea and acne vulgaris of moderate degree. The finger nails were rather flattened and were brittle. There was puffiness of the eyelids. The urine showed no abnormal constituent. She gave a history of dyspnoea and palpitation on exertion. Her pulse rate was as follows:-

Sitting .....	90
Standing .....	110
after exercise ...	140, and took three minutes to

return to the region of 110. The blood pressure was 110/

110/74 mm. mercury. There was no sign of an enlarged thyroid gland; no eye changes of exophthalmic goitre, and no fine tremor of the fingers or eyelids. The skin was not moist, but greasy as noted above. There were no nipple changes or other signs of pregnancy. The heart sounds themselves were pure and closed in all areas. I could detect no abnormality in the lungs.

I examined her blood and found the following:-

12.11.37.

Haemoglobin.	64.
R.B.C.	4.500.000.
W.B.C.	6.800.
C.I.	0.71.

I then sent her over to the Royal Infirmary of Edinburgh where it was found that there was no radiological evidence of tuberculosis. I prescribed Ferri. et. ammon. cit. gr. xx, t.i.d. and after one week increased it to Q.I.D. This was tolerated quite well with no untoward effect. After a third week the dose was increased to gr. xxx, t.i.d. and after ten days the patient began to feel very much better. Her appetite improved and she tolerated the large dose quite well. She felt much fitter, but was still breathless on exertion. Her pulse after walking up a very steep hill to the surgery was in the region of 90-100. The blood examination approximately two months later showed a marked improvement in the haemoglobin level, viz.

19.1.38./



19.1.38.

Haemoglobin.

83.

R.B.C.

4.670.000.

C.I.

0.90.

She was advised to continue Ferri. et. ammon. cit. gr. xxx, t.i.d.

4.2.38.

Haemoglobin.

92.

The menstrual periods are now coming approximately every 5 weeks and last only 3 days. The loss is scanty, but the menses are more regular.

The pulse, however, is not yet down to a satisfactory level and remains in the region of 85 - 95. There is a moderately accentuated sinus arrhythmia but even allowing for this, the patient still presents a slight tachycardia.

She is working now and appears to be none the worse of her exertion. She reports every two weeks.

Case 24. Nellie N. aet 21.

This girl works as a clerkess in the local post-office. Unfortunately, she was not my patient, so my notes on her are scanty. She had consulted my principal two months previously for acne vulgaris, which he had treated successfully with an autogenous vaccine. In the course of routine examination he discovered that she had a tachycardia and puffy ankles. He prescribed Ferrous sulphate gr. iiii, t.i.d. and she made such a striking improvement that he asked me, as a point of interest, to do a blood count. She had been taking/

taking the Ferrous sulphate pill for six weeks before I could arrange to have this done. and the result was as follows:-

Haemoglobin.	100.
R.B.C.	5.800.000.
C.I.	0.86.
Halometer reading.	6.9 u.

Case 25. Sadie C. aet 21.

This girl is an apprentice to the local chemist. Her father died several years ago. Her mother, who died of a cerebral haemorrhage five months ago, was a chronic invalid with essential hypertension. Her blood pressure was always in the region of 200/130 mm. mercury, and she had severe intractable asthma. In her later years Mrs. C. was more or less confined to bed and when allowed up was unfit for routine house-work. She had two sons aged 19 and 23. The cooking and most of the house-work had to be done by Sadie, fitted in between her hours on duty at the shop. The family resources could not afford a help. The meals were, perforce, the type that could be quickly, easily, and cheaply prepared. Mince, corned beef, or pies, tea and buns, or scrambled eggs, were usually the chief course of the main meal of the day. Low roughage, low vitamin, poor class protein, and indifferent cooking, characterised these meals, which were usually eaten, very hurriedly, in a dingy little two-roomed house. The larger room served as a kitchen, dining and sitting room, and bathroom/

bathroom - the only tap and sink were in this room - as well as bedroom for Sadie and her mother at night.

Sadie herself had been the subject of acute rheumatic fever in childhood and had a well marked mitral stenosis.

She was of the thin, anxious "Cassius" type. She said she had always been "bloodless" and for the past five years had attended the doctor regularly at intervals for a tonic. She took Ferri. et ammon. cit. gr. xv, t.i.d. for 6 - 8 weeks and felt considerably improved. She would then lapse back again in 2 - 3 months' time.

There was no history of menorrhagia or bleeding haemorrhoids.

The menarche occurred at the age of 14 and the periods have been quite regular and apparently normal since.

I examined her blood two months after her last course of iron and found the following:-

8.12.36.

Haemoglobin.	74.
R.B.C.	4.530.000.
C.I.	0.82.
Halometer reading.	7.0 u.

Case 26. Mrs. M. aet 22.

Nulliparous.

Menarche occurred at the age of 16 and the periods have been irregular since. This patient lives with her mother/

mother (her husband is at sea) and sister, in a four-roomed house. She is a lady of uncertain habits and addictions. Although she has a comfortable income for her station in life, her dietary arrangements are carried out in a haphazard, squalid, and most unsatisfactory manner. She was advised, several years ago, to try gin as a cure for her dysmenorrhoea. The dysmenorrhoea continues unabated and, unfortunately, the gin cup is still in active service.

I first saw her in October, 1936. She gave a history of weakness, dizziness, palpitations, dysmenorrhoea, profuse menorrhagia, and metrorrhagia, of five years' duration. She had a dilatation and curettage done 3 years ago with considerable relief. The symptoms returned, however, in one year's time.

On examination I discovered that she had a tachycardia, and it was noticeable that she had a marked swelling of the thyroid gland. The eyes were somewhat prominent, and there was a lag in the downward descent of the upper lid. I sent her over to the Royal Infirmary, where she was seen by a gynaecologist who found that she had a severe chronic cervicitis, which required operative treatment. A surgeon arranged to admit her after her pelvic condition had been dealt with.

I examined her blood and found the following:-

1.11.36/



1.11.36.

Harmoglobin.	84.
R.B.C.	4.960.000.
C.I.	0.86.
Halometer reading.	6.9.

I prescribed Ferrous sulphate gr. iii, t.i.d. She did not report to me and I have since heard that she has left the district.

The case illustrates many interesting points which will be more fully indicated below.

Case 27. Mrs D. aet 23.

Para 2.

This woman lives in a two-roomed house. Her husband earns on an average £2.15/- per week. The average income is not nearly so low as in some other cases quoted in this paper.

Mr D. asked me to call on his wife as she was "badly needing a tonic". Mrs D. when I called on her, was busily engaged trying to keep an eye on her two children - aged 7 months and 2 years respectively - cook a dinner, and do the week's washing, all at one time.

It was noticed that she was very pale. Many of the other cases mentioned had a pale face but Mrs D's pallor was of a different nature. She looked, literally, bloodless. Her skin had a dead lustreless appearance, most difficult to describe. She had a wan, weary expression. The mucous membranes of the eyes, lips/

lips, gums, and palate were blanched. Her finger nails were flattened, brittle and cracked. I could detect no abnormality in the tongue, but she complained of difficulty in swallowing. Her pulse was 105, and her blood pressure 105/70. She had a soft, blowing systolic murmur at the apex, but no murmurs could be detected at the base of the heart or in the regions of the great vessels.

The menarche occurred at the age of 15. The periods had been irregular, occurring every 5 to 6 weeks and lasting only six days. Mrs D. said the loss was very slight, only faintly soiling her diaper. She had had one period since the birth of her last baby, who was now weaned.

Her leading symptoms in the order in which she gave them were, loss of energy, disinclination for food dysphagia, and amenorrhoea.

The last mentioned was uppermost in her mind. Her menstrual period was one month overdue - she had had one period since the birth of her last baby - and the possibility of a third pregnancy with all that it would entail, was causing her acute anxiety and distress.

I enquired about her diet and found that she did not like cooking. Tea, pastry, condensed milk, rolls, bread and margarine, was the menu for at least two meals per day. Kippers, sausages, and occasionally bacon/

bacon, were had twice or three times a week for tea, always fried. Dinner consisted of mince, boiled mutton, or beef with large helpings of potatoes, on three days a week. There was no dinner on Sunday - a late breakfast fry obviating the necessity for a mid-day meal. Washing-day's dinner consisted of porridge and a fry. Soup was taken on the days after the boiled mutton or beef, and was reheated to serve two days. The turnip, cabbage and carrot were usually boiled in the soup for hours on end, and not cooked separately, in order to save time and fuel cost.

The examination of the blood yielded the following results:-

15.11.36.

Haemoglobin.	45.
R.B.C.	4.050.000.
C.I.	0.56.
W.B.C.	7.100.
Halometer reading.	7.0 u.

I prescribed Ferrous sulphate gr. iiii, t.i.d. and suggested that she take an orange per day.

She reported three weeks after starting iron therapy and there seemed to be a definite improvement. She said that she was feeling stronger, but complained that the pills were giving her heartburn. I prescribed acid. hydrochlor. dil. B.P. 3 i, to be taken, well diluted, with meals.

She came back one week later in radiant spirits. Her menstrual period had commenced. The digestive upset was much less. I saw her six weeks after commencing/

commencing iron therapy. There was no dysphagia; the digestive disturbances were practically negligible; the appetite, she said, was markedly improved, and she felt more fit for her work.

How much of the improvement was of psychologic origin I cannot say. Certainly the digestion and appetite were improved, and there was no tachycardia. The exercise tolerance test gave a more satisfactory result.

Pulse, sitting.....80  
standing.....88  
after exercise.....120, returning to 90

after  $1\frac{1}{2}$  minutes.

She had taken 160 pills of Ferrous sulphate gr.iii. She was to report in three days for a blood count but failed to do so. She lapsed from benefit in the medical club of which she was a member, and passed from my ken.

I saw her fortuitously fifteen months later when I called in to see her husband, who had joined my principal's panel. She said she had felt much better after her last attendance on me and thought she was so much improved that she did not need a doctor.

In June of 1937 she had felt "run-down" and had taken the iron pills for one month. (The prescription for 100 pills were repeated by the chemist).

I examined her and found that her general condition/



condition was much improved. There was no dysphagia. The nails were still brittle but improved. The appetite was fairly good but she was troubled by occasional heartburn and flatulence. She did her own cooking and general housework. Her dietary arrangements were, I am afraid, but little changed.

Her periods were more regular, being **never** more than one week overdue. Her pulse rate was 82 per minute. Examination of her blood (which was achieved after much bullying and moral persuasion) revealed the following:-

<u>16.2.38.</u>	
Haemoglobin.	77.
R.B.C.	5.260.000.
C.I.	0.74.
Halometer reading.	

Apart from the many points of clinical interest, which are discussed in another section, this case illustrates a few of the difficulties with which one meets, in attempting to obtain scientifically controlled data and adequate progress and follow-up notes, under general practice conditions.

#### Case 28. Nurse R. aet 24.

This patient is an acting sister in a mental institution a few miles away. Her home is nearby and she consulted me one day while on leave. She gave me a history of having fainted two weeks ago, and of feeling run-down, easily tired, and generally unfit for her rather arduous duties. The menarche occurred at the age of 14 and the menstrual periods were regular and/

and apparently normal.

She looked rather pale, and on examination, it was noted that the mucous membranes were pale, but not unduly so. The pulse rate was 82 per minute and she responded satisfactorily to the exercise tolerance test. The heart sounds were closed and pure. There was no evidence of any nail changes or indication of any dysphagia or digestive upset. There was nothing else of note in the clinical findings.

I examined the blood and found the following:-

17.10.37.

Haemoglobin.	69.
R.B.C.	4.240.000.
C.I.	0.82.
W.B.C.	7.200.
Halometer reading.	7.4 u.

I prescribed Ferrous sulphate gr. iii, t.i.d.

She tolerated this very well, and had no digestive upset. When I saw her five weeks later she was looking and feeling very much improved. Her skin had a healthy glow, the pulse was 74 and of good volume. The appetite had improved greatly. She felt quite fit for her work and was very enthusiastic about the iron therapy.

The haematological findings were:-

25.11.37.

Haemoglobin.	91.
R.B.C.	5.220.000.
C.I.	0.88.
Halometer reading.	7.2 u.

On enquiry as to her daily menu I found that she had been eating very little when I first saw her - she/

she complained about the cooking, and method of serving of her meals at the hospital. On the second occasion she admitted that the quality and quantity of the food, while far from being perfect, were of a reasonably high standard. She said too, that she was eating more, and enjoying her meals better after the commencement of iron therapy.

Case 29. Mrs P. aet 24.

Para 1.

This young wife had one baby of ten months. She was living in a three-roomed house with her father and mother, and three sisters. Her husband was in the Royal Air Force, and stationed in England. Mrs P. had no complaints. I met her when I was called to treat the baby who had "the hives." She was a blonde, pale-faced woman, who told me she had always been "bloodless." The menarche occurred at the age of 17, and the periods had been scanty and irregular since, always associated more or less with pain. There was pallor of the conjunctivae of a moderate degree, but nothing else of note.

The examination of the blood revealed the following:-

28.11.36.

Haemoglobin.	84.
R.B.C.	5.960.000.
C.I.	0.71.
Halometer reading	6.8 u.

I/



I prescribed Ferrous sulphate gr. iii, t.i.d. and she shortly afterwards left to join her husband. I have not seen her since.

She made a very dramatic improvement.

Case 30. Mrs R. aet 25. (She walked up a steep hill

Para 1. to see me) she had been taking four

This was an extremely pale woman. She gave me a history of having been in good health until the birth of her baby ten months previously. She had no doctor at her confinement and the nurse informed her that she had had quite a brisk post partum haemorrhage. She had a very stormy puerperium and was confined to bed for two months after the birth of her child.

My principal saw her eight months after her confinement when he was called in to treat her for an attack of severe vomiting.

She was collapsed. Her pulse rate was 140 per minute in bed, and she could not raise her head from the pillow without fainting. She had had only one period since the birth of her child and she had at that time, a very slight blood-stained vaginal discharge. It was thought at first that she was a case of ruptured tubal pregnancy, but she improved with vigorous symptomatic treatment directed towards the alleviation of the shock, viz. warmth, fluids, glucose, etc.

Unfortunately a blood count was not done at this time./



time. When the stomach settled, iron was cautiously introduced in the form of Ferrous sulphate three grain pills - one, two, three, and finally four pills daily.

She made a very dramatic improvement.

When I first saw her (she walked up a steep hill to the surgery to see me!) she had been taking four three grain pills daily for about eight weeks. She was in very good spirits, and spoke very warmly of my principal, who, she declared, had "saved her life and made an entirely new woman out of her."

She said she was quite fit for her household duties. She lived in a two-roomed house with her baby and husband, who was stationed at the local aerodrome with the Royal Air Force. She did her weekly washing unassisted, although she felt rather tired after it, and was a little short of breath when she climbed a hill or stairs.

She looked very pale, the mucous membranes were pale, and the nails were flattened and brittle. The pulse was regular, the rate being 96 per minute. There was a faint systolic murmur at the mitral and pulmonary areas. The spleen was not enlarged and there was nothing else to be made out by physical examination.

The blood examination revealed the following:-

24.10.37.

Haemoglobin.	65.
R.B.C.	4.250.000.
C.I.	0.77.
W.B.C.	5.800.000.
Halometer reading.	7.0 u.

She/

She was advised to continue taking the Ferrous sulphate gr. iii, Q.I.D. One month later her figures were as follows:-

24.11.37.

Haemoglobin. 88.

I met her fortuitously two months later. She was still taking her pills regularly - three to four a day. I induced her to have a further check up and the haematological findings were as follows:-

30.1.38.

Haemoglobin.	91.
R.B.C.	5.190.000.
C.I.	0.89.
W.B.C.	6.000.

She was advised to take one pill three times a day for one month, stop one month, and then report.

Most of my patients were extremely grateful for the rapid clinical improvement which followed the exhibition of iron therapy, but she was one of the few, who of their own accord, continued to take the pills long after the improvement was established.

A great many of the cases, as will be evidenced below, not only ceased therapy as soon as the clinical improvement was established (the appearance of a belated menstrual period, the return of appetite, the absence of lassitude) but they also failed to report at all. The checking up of these cases under general practice conditions is a matter of extreme difficulty. Even when the patients have been rounded up, they may (although I have found this the exception rather than the rule) adopt the attitude of "conscientious objection."

Case 31. Mrs Mary C. aet 27.

Mrs C. came to me complaining of amenorrhoea.

Her/

Her reproductive history was as follows:-

The menarche occurred at the age of  $14\frac{1}{2}$ . The periods, at first irregular, settled into a regular cycle and continued so till her marriage at the age of 21. She had two children aged six and four years. Both confinements appeared to be perfectly normal, although she complained that they "took a great deal out of her." The periods became more frequent after the birth of the last baby, and for the last three years she had had menorrhagia and metrorrhagia - the periods coming, on an average, every three weeks, and lasting eight to ten days. She had not had a period for seven weeks when I examined her and she suspected a pregnancy.

With regard to her general medical history, the following was noted. She had rheumatic fever while a school child. This was of the classical polyarticular type, flitting from joint to joint. There was evidently some cardiac damage at that time for she was confined to bed for two months. For the past five years she had been troubled at irregular intervals with "rheumatism" of various parts of the body. In particular the small joints of the fingers, the wrists, the elbows, and neck, had been the sites of painful arthritis. Recently she noticed a creaking in the tempero-mandibular joint when the mouth was opened.

On the day I first examined her the leading symptoms,/

symptoms, apart from the amenorrhoea and painfully stiff joints mentioned above, were weariness, breathlessness, and palpitation on exertion, of one year's duration; disinclination, amounting almost to a dislike, for food for the past two years; a feeling of heaviness after eating for the past four months; dysphagia during the previous two months; insomnia of one year's duration; and swelling of the ankles over the same period.

On physical examination the following was noted. She was a small thin woman, with a pale, muddy complexion. She had dull, lustreless eyes, a gaping mouth, and a wan, docile, almost stupid, expression.

Attention was first of all directed to her joints. Several of the metacarpo-phalangeal, two inter-phalangeal, and both wrist joints were swollen by peri-articular proliferation. In passing it was noticed that the finger nails were concave, and many of them cracked and ridged. They were the nearest approach I have, personally, seen to the text-book illustrations of koilonychia. Creaking was elicited in the cervical intervertebral joints, and in both temporomandibular joints.

The pulse was rapid (120 per minute) and irregularly irregular. The irregularities, because of their comparatively slow frequencies, I put down as extra systoles. There was a definite systolic thrill on/



on palpation over the mitral area. On auscultation a well marked rough presystolic murmur propagated into the axilla, was noted, and the diagnosis of premature systole's confirmed. There was a markedly accentuated second sound in the pulmonary area.

There was no enlargement of the liver or spleen. The mouth was next examined. There were fifteen markedly carious teeth. The tonsils were small, shrunken and pitted. There was marked pallor of the mucous membranes of the gums, lips, and palate. The tongue showed a certain amount of papillary atrophy at the edges - especially the left. It was not sore.

Attention was next directed towards the patient's complaint of swollen feet. Both ankles showed pitting on pressure, an oedema of modest degree.

The following day she reported with a specimen of her urine, which had no abnormal constituent. The blood was examined on that day and the following noted:-

1.11.36.

Haemoglobin.	56.
R.B.C.	4.860.000.
C.I.	0.58.
Halometer reading.	6.8 u.

I prescribed Ferrous sulphate gr. vi, t.i.d. She reported in one week complaining of indigestion. She was given ac. hydrochlor. dil. 3 ii, t.i.d., well diluted, with meals.

She reported two weeks later and cascara was prescribed for severe constipation. There was frequency/

frequency of micturition but no morning sickness. She was given linament of methyl salicylate as an external application for her joints.

She was tolerating the pills quite well and her digestive symptoms were relieved. The appetite had improved in ten days, and after two weeks she said she felt markedly improved.

I thought most of the improvement must be psychological in origin because of the short duration of the therapy but 35 days after the commencement of iron the blood showed the following:-

5.12.36.

Haemoglobin.	88.
R.B.C.	5.520.000.
C.I.	0.80.
Halometer reading.	7.2 u.

Though there were no positive signs I felt certain that she was now pregnant, and she herself accepted the inevitable with the placid (and, I often think, courageous) calm which characterises those of her class.

The diagnosis of pregnancy was established one month later.

The rheumatoid condition subsided and her general condition was enormously improved.

Throughout January, February, and March, 1937, she continued taking Ferrous sulphate gr. iii, t.i.d. The dysphagia had gone in four weeks. There was no breathlessness/

breathlessness except on severe exertion. There was no oedema. The appetite was greatly improved and the insomnia relieved. The cardiac condition appeared to be compensated and the pulse rate was 82 per minute and regular. A feature which struck me particularly was the change in this woman's mental activity. She became more alert, vivacious, and at times even witty.

Towards the end of March she began to show signs of cardiac failure, oedema of the ankles, tachycardia, and, finally, auricular fibrillation. She was now seven months pregnant. She was put on Tincture of Digitalis M xv, q.i.d. and with rest in bed was beginning to improve when I left the district in April to take up a hospital appointment. I returned to the practice in September, and learned that she was delivered of a healthy baby, spontaneously, in June.

She stopped taking the pills during the 9th month but took them for one month after the birth of her child (which, incidentally, was breast fed). She said that this was the easiest of all her confinements. She was kept on a maintenance dose of Digitalis M x, t.i.d. for three months after her confinement.

I saw her again in October 1937. Her general condition was good but the joints were rather painful. I prescribed iron salicylate in the following form.

Liq./

Liq. Ferri. Perchlor.	M x.
Sod. salicylate.	
Pot. bicarb. aa	gr.x.
Liq. strych. hydrochlor.	M iii.
Glycerini.	M x.
Aqua ad.	3 ii.
3 ii, ex aqua, t.i.d. p.c.	

She took this for one month with considerable relief. She had no further Digitalis. She refused to have her teeth out without a general anaesthetic, and this could not be done for medical and financial reasons. (I feared the results of stirring up sepsis by a whole-sale extraction, and, on the other hand, her cardiac condition and financial state contra-indicated repeated administrations of a general anaesthetic).

She went for a short holiday in November and was much improved.

I saw her in December, 1937, and advised her to take a course of Ferrous sulphate gr. iii, t.i.d. At the end of this course she was, at last, persuaded to have her teeth out - two or three at a time, at intervals of five to seven days. She had had five extractions at the end of January 1938, and I examined her blood to find the following:-

31.1.38.

Haemoglobin.	84.
R.B.C.	5.020.000.
C.I.	0.84.

The dietary arrangements were practically similar to those of Mrs D. (case 27.).

Case 32. Annie D. aet 28.

This/



This patient was a domestic servant. She had been treated for ten weeks before I saw her and was taking Ferri. et. ammon. cit. gr. xv, t.i.d. She gave a history as follows.

She said she had always been pale and "bloodless." Her periods began at the age of 17. They were at first scanty and irregular, but became more regular later. In the three months before treatment commenced she had been greatly troubled with polymenorrhoea and metrorrhagia. The loss was marked and she felt that menstruation took "a great deal out of her." She was very easily tired and had been troubled with insomnia for one year.

Her ankles were swollen at the end of each day's work. She said that her appetite was poor and that she was troubled with flatulence, indigestion, and constipation. The latter had been very severe on her dose of iron (Ferri. et. ammon. cit. gr. xv, t.i.d.) and required the regular use of an aperient.

On examination I found that she was very pale. There was moderate pallor of the mucous membranes. There were no tongue or nail changes.

In answer to a leading question she said that she had had difficulty in swallowing for the past six months but it was now gone.

The pulse was regular and rapid (110 per minute). The cardiac sounds were closed and pure. There was no oedema/

oedema of the face or ankles. The examination of the blood revealed the following:-

17.10.37.

Haemoglobin.	63.
R.B.C.	4.020 000
C.I.	0.79.
Halometer reading.	7.4 u.

The patient said she had felt much better since she commenced iron therapy ten weeks previously.

I prescribed Ferri. et. ammon. cit. gr. xxv, t.i.d.

but she left the district shortly afterwards and I have not seen her since.

Case 33. Helen W. aet 28.

This patient is a cook. She works in the same establishment as Mrs Jessie T., case 39.

The menarche occurred at the age of  $15\frac{1}{2}$ . The periods were at first regular, but at the time of consultation she complained of menorrhagia. The periods came every three weeks - lasting about eight days with a loss which was, in her opinion, greater than normal.

She gave a history of gradually increasing loss of energy, lack of appetite, inability to concentrate, palpitation and breathlessness on exertion, over a period of one year. The lack of appetite amounted almost to a distaste for food. She said that she felt her food "stick in her throat." (Dysphagia ?)

On examination it was noted that she was a pale-faced woman of slender build. The mucous membranes of the eyes, lips, gums and palate were pale. The tongue/

tongue was apparently normal. There was no oedema of the face or hands. The nails were very brittle with irregular cracked edges.

The pulse was rapid (120 per minute) and regular. After exercise the patient felt conscious of palpitation but no irregularity of the pulse was observed. There was a systolic murmur in the pulmonary and aortic areas.

The blood was examined and the following noted:-

30.10.36.

Haemoglobin.	60.
R.B.C.	3.800.000.
C.I.	0.79.
Halometer reading.	7.3 u.

I prescribed Ferri. et. ammon. cit. gr. xv, t.i.d. I saw her two weeks later and she was feeling much better.

After a period of two weeks she looked much better and the pulse had dropped to 90 - 100 per minute. She was less breathless on exertion. She continued on this dose of scale iron till January 1937, when her blood was again examined and the following noted:-

10.1.37.

Haemoglobin.	82.
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Two weeks later this patient was a prey to a severe attack of influenza, which had reached epidemic incidence locally at this time. Immediately her temperature settled she continued with her iron - Ferri. et. ammon cit. gr. xv, t.i.d. Her blood was examined and the following noted:-

7.2.37./

7.2.37.

Haemoglobin.	83.
R.B.C.	4.720.000.
C.I.	0.88.
Halometer reading.	7.3 u.

She was prescribed saccharated ferrous carbonate in bulk, and advised to take  $\frac{1}{2}$  teaspoonful three times a day. This she continued to do for four months.

I next saw her in September, 1937, when I returned to the district. She had taken no medicinal iron for the preceding three months. I checked the haemoglobin level of the blood and found the following.

1.10.37.

Haemoglobin.	82.
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I prescribed Glaxo. Ferrous sulphate gr. iiii, t.i.d. and three weeks later found the following:-

23.10.37.

Haemoglobin.	108.
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This patient (and her friend - case 39) was a domestic servant in the establishment of one of the wealthiest families in the district. Her diet, however, was very inadequate in this rather strictly-run menage. The diet was deficient in proteins and vitamins, and iron-rich foodstuffs. There was no fresh fruit available. Vegetables were strictly rationed. When one considers that this patient spent most of her day preparing and cooking meals, and that she had a marked anorexia, and a certain degree of dysphagia, one is justified in postulating a deficient intake of iron. Deficient intake of a diet itself deficient in the factors necessary for efficient haemopoiesis/



haemopoiesis, sets in motion a vicious circle, which can only be broken by the exhibition of some iron preparation, in adequate dosage, and over an adequate period of time.

Case 34. Mrs K. aet 28.

This patient is the mother of three children. She lives in one of the many houses which have been condemned by the local sanitary authorities. Her husband earns 37/6d per week at the local dockyard when he is employed. He is unemployed for about six or seven months of the year. I delivered her of her third child in October, 1936. There was nothing pathological about the birth or puerperium and she made satisfactory progress. I had ample opportunity, while attending her, to observe the domestic arrangements. The diet was very similar to that of a great many families in the district and was on the lines of the S- family, mentioned in case 12.

Mrs K. looked very pale. The mucous membranes were definitely pale. There was no tachycardia or oedema. Apart from the pallor there was no evidence of anaemia. With the exception of an anorexia of moderate degree and an occasional bout of flatulent dyspepsia she had no other complaints, and presented a surprisingly happy and cheerful countenance in the face of such domestic hardships. The examination of/

of her blood three weeks after the birth of her child revealed the following:-

28.11.36.

Haemoglobin.	77.
R.B.C.	4.880.000.
C.I.	0.80.
Halometer reading.	7.0 u.

I prescribed Glaxo. Ferrous. sulphate gr. iii, t.i.d. She reported two weeks later. The digestive symptoms were greatly relieved and she was feeling much better. She failed to report again.

Case 35. Nan H. aet 28.

This patient was a highly nervous, mentally unbalanced woman. A confirmed spinster she had no brothers or sisters, and lived with her father and aunt, for whom she was a continual source of anxiety and apprehension. She consulted me for treatment of chilblains. I discovered that she suffered from metropathia haemorrhagica of severe degree. From her aunt I discovered the sexual history of this woman was most unsatisfactory. The periods did not commence till the age of 19 and, at first scanty and irregular, had gradually become worse and associated with severe dysmenorrhoea. There was, obviously, some underlying endocrine lack, which must have to some extent, accounted for the mental aberrations.

Examination revealed no evidence of anaemia, but in view of the menorrhagia I decided, as a point of interest/

interest, to examine the blood and found the following:-

4.11.36.

Haemoglobin.	91.
R.B.C.	4.740.000.
C.I.	0.97.
Halometer reading.	7.5 u.

I prescribed Ferri. et. ammon. cit. gr. xxx, t.i.d. and examined the blood after three weeks to discover the following:-

23.11.36.

Haemoglobin.	85.
R.B.C.	4.470.000.
C.I.	0.97.
Halometer reading.	7.2 u.

The menorrhagia continued unabated and she was seen in consultation by a gynaecologist, who advised induction of menopause.

This was done by the exhibition of deep X-rays, and the patient had considerable relief.

I saw her one year later. There was no change in the mental condition.

Case 36. Isobel F. aet 29.

This patient was a shop assistant who had just recovered from an attack of gastric influenza, complicated by a slight degree of jaundice. Her gastric condition rapidly improved with the exhibition of fractional doses of calomel followed by a saline aperient; a suitable diet was prescribed. She had no bradycardia, but manifested, rather, a fairly marked degree of tachycardia throughout the illness and convalescence./

convalescence. The pulse continued for three weeks in the region of 100 - 110, and was characterised by a fairly well marked sinus arrhythmia. The cardiac sounds were closed and pure. There was no evidence of pulmonary mischief.

The mucous membranes showed a moderate degree of pallor. Apart from this, the physical examination revealed no positive finding. I examined the blood to find the following:-

3.12.37.

Haemoglobin.	103.
R.B.C.	5.480.000.
C.I.	0.95.
Halometer reading.	6.8 u.
W.B.C.	5.800.

I prescribed Ferri. et. ammon. cit. gr. xx, t.i.d in water, as a "tonic," and advised her to lead a restricted life. In two weeks the pulse was in the region of 80 and she was feeling quite fit for her work to which she returned.

There was a definite functional element at play in the production of this tachycardia. Whereas due allowance must be made for the powers of suggestion, I am loath to relinquish the impression, however illogical, that the simple exhibition of scale iron alone in water, did (as in many other personal cases not quoted in this paper) act as a tonic in this case which was nevertheless not demonstrably anaemic.

Case 37. Mrs Jessie F. aet 29.

This patient was a domestic servant. She had had one child which died at birth. She consulted me on account of "palpitations" which she said, had troubled/



troubled her intermittently for about two years, but had become much worse in the past three months. She gave a history of having been "bloodless" as long as she could remember. For the past five years she had been breathless on exertion and she noticed her heart thumping if she did any heavy work. She had been troubled for two years with indigestion, loss of appetite amounting almost to a distaste for food, and a feeling of heaviness after eating. She had had difficulty in swallowing for one year but this varied in severity. She said she was very nervous and often felt as if she were "going to choke." Her memory was poor and she had suffered from insomnia for three months. Her periods were scanty and irregular; she would often pass two, and sometimes three or four months without menstruating. She complained of sore feet and said her ankles were sometimes swollen after a specially hard day's work.

She acted as an occasional charlady, and managed her own household duties as well. Her husband who was a chauffeur, earned £2.5/- per week. There was no actual poverty in the house but her diet was deficient in fresh fruit and vegetables, and contained an unnecessary high proportion of fried and greasy dishes. She was very fond of chocolates and sweets.

On examination it was observed that she was a very pale faced woman with a dull, lustreless, expression. The mucous membranes were blanched. She had no/

no tongue changes, but had several carious teeth. The finger nails were spoon-shaped and brittle. There was no oedema of the face or legs. The pulse rate was 110 per minute and the rhythm was irregular. The heart sounds were closed and pure in all areas, but extra systoles occurred every ten or twelve beats - an average of 10 to 15 per minute. There was no splenic enlargement. The blood was examined and the following noted:-

7.12.36.

Haemoglobin.	38.
R.B.C.	2.820.000.
C.I.	0.68.
Halometer reading.	6.7 u.

On direct questioning it was found that this patient suffered from obstinate constipation, and that she had prolapsed haemorrhoids which occasionally bled profusely.

Ferrous sulphate gr. iii, was prescribed, and in three weeks the patient was very much improved.

The dyspnoea on exertion had gone, the appetite had improved, and the dysphagia was scarcely noticeable (she had no difficulty in swallowing the pills).

There was a general sense of increased well being.

Unfortunately this patient shortly afterwards left the district and I have not seen her since.

Case 38. Alex. McL. aet 30.

This patient was one of the local police constables/

constables. I had attended him for a pharyngitis and arranged to examine his blood as a control, two weeks after he recovered from his illness. At the time of examination the conjunctivae were pale but nothing else was noted.

The blood picture was:-

18.2.37.

Haemoglobin.	107.
R.B.C.	5.500.000.
C.I.	0.99.

Case 39. Mrs Jessie T.

This patient was a married nulliparous woman, who was engaged as a domestic servant in the same establishment as Helen W. (case 33).

I was called in to see her for the first time during an attack of acute tonsillitis, from which she made a rapid and uneventful recovery. I noticed that she looked very pale and that the conjunctivae were pale. Her pulse remained in the region of 90 after the nasopharyngeal condition had settled. She received no iron therapy after the tonsillitis. I asked her to report to me in four weeks.

On examination it was noted that the pallor had not abated and the pulse had not settled. There were no tongue or nail changes, but there was a history of flatulent dyspepsia and a slight degree of dysphagia antecedent to the tonsillitis, over a period of approximately one year. She had dyspnoea on moderate exertion/

exertion. The periods were scanty and had been missing for nine weeks. I could elicit no other symptoms or signs of pregnancy. A blood examination revealed the following:-

21.10.37.

Haemoglobin.	49.
R.B.C.	4.010.000.
C.I.	0.61.
Halometer reading.	6.7 u.
W.B.C.	6.600.

I prescribed Ferrous sulphate gr. iii, t.i.d.

She reported in ten days feeling already much improved in her general health. She had had a menstrual period and she said that her appetite was improving although the pills had caused a certain degree of dyspepsia at first. The blood picture four weeks after the commencement of therapy, was:-

20.11.37.

Haemoglobin.	86.
R.B.C.	5.040.000.
C.I.	0.86.
Halometer reading.	7.1 u.

She was married shortly after-wards and left the district. She came to see me, however, at frequent intervals for a period of three months, during which time she continued to take Ferrous sulphate gr. iii, and made a very dramatic improvement.

As she could only call on me in the evenings I could not estimate her haemoglobin level (as I used a Sahli's instrument) but although I have no further blood figures her marked clinical improvement was, to my mind, and her's, ample justification for the therapy exhibited./



exhibited.

Case 40. Mrs M. aet 32.

This patient is the wife of a local tradesman and lives with her only child (case 13) in a comfortable home. She had a small subcutaneous whitlow, the result of a prick from a needle, which I incised. It was quite a trivial matter and cleared up in three or four days. She herself suggested (as so many patients do, who are the subjects of acne vulgaris, farunculosis, "heat spots" and similar skin conditions) that her "blood was out of order." She said she had been "off colour and out of sorts" for several weeks. She was rather easily tired, was rather irritable, and did not feel fit for her household duties. Her appetite was slightly impaired. She felt she should have a "tonic."

Clinical examination revealed no evidence of organic disease. She had a rosy, healthy complexion, and the mucous membranes could not be accurately described as being even moderately pale.

There were a few small ulcers along the edge of the tongue and the whole organ looked rather red. There were no nail changes.

I examined the blood and found the following:-

25.10.36.

Haemoglobin.	74.
R.B.C.	4.910.000.
C.I.	0.76.
Halometer reading.	7.0 u.

I prescribed Glaxo. Ferrous sulphate gr. iii, t.i.d. and advised her to take an orange at least every second day.

She took 100 tablets and when I saw her at the end of one month, she was looking and feeling much happier. She said she was enormously improved, her appetite was better, and she was most enthusiastic about the iron therapy which she felt had "done her a lot of good."

She felt very fit and was taking a very active interest in local social activities.

I examined the blood and the following results were noted.

5.12.36.

Haemoglobin.	101.
R.B.C.	5.380.000.
C.I.	0.95.
Halometer reading.	7.3 u.

This case is, to my mind, a very striking example of the marked improvement in general well being which frequently follows the exhibition of iron therapy in a case of anaemia of only moderate degree.

The patient's complaints were extremely vague, and there was, in point of fact, no certain criteria on which to base a diagnosis of anaemia other than the results of the blood examination.

Case 41. Mrs A. aet 33.

This patient is a daughter of Mrs. M. (case      ). She is married and has one child - a girl aet 5. She was a tall, heavily-built woman, who consulted me on account of amenorrhoea. She said she felt very well after the birth of her only child, but for the past year had been vaguely "out of sorts" and easily tired. She occasionally felt so "fagged out" at the end of a day's work that she would weep. She was breathless on moderate exertion. She said her ankles were swollen after an especially hard day - e.g. after washing. She said that she had experienced difficulty in swallowing for the past six months. Her husband earned only 38/- per week on an average, and as she and her daughter were always very smartly dressed I am afraid her dietary expenditure was insufficient to supply the numerous physiological requirements.

On examination I observed that she was a pale-faced woman. The mucous membranes were noticeably pale. There were a few minute ulcers on the edge of the tongue, and on direct questioning the patient stated that she had a sore tongue which she put down to a jagged tooth. There were no nail changes. There was no evidence of organic disease of the cardiovascular system. The pulse rate was 84 per minute. I could discover no other positive finding. I examined the blood/

blood and found the following:-

1.11.36.

Haemoglobin.	67.
R.B.C.	4.250.000.
C.I.	0.81.
Halometer reading.	7.0 u.

I prescribed Glaxo. Ferrous sulphate gr. iii, t.i.d. and after ten days there was a noticeable improvement. After only two weeks the patient was very enthusiastic about the treatment adopted. The appetite was improved. The dysphagia had gone. The dyspnoea was much less. After one week more her belated period arrived and the blood was again examined.

5.12.36.

Haemoglobin.	80.
R.B.C.	4.200000.
C.I.	0.95.
Halometer reading.	7.0 u.

She continued taking the Ferrous sulphate pills for three months after many exhortations and admonitions on my part. The menstrual cycle was established in a regular rhythm, and the patient was apparently in perfect health when I examined her after three months to find the following:-

4.3.37.

Haemoglobin.	94.
R.B.C.	4.730.000.
C.I.	1.00.
Halometer reading.	7.3 u.

I did not see her again for eight months. In October 1937 when I saw her she confessed that she had ceased iron therapy in April. She had had a period of amenorrhoea/



amenorrhoea in June - July. At the time of examination she was vaguely "out of sorts" and felt easily tired.

I estimated the haemoglobin and found it to be:-

10.10.37.

Haemoglobin.

78.

I again prescribed Ferrous sulphate which she took for two months. The blood on January 1938 was as follows:-

28.1.38.

Haemoglobin.

98.

Case 42. Mrs W. aet 33.

This patient, whose husband is a petty officer in the Navy, lives with her only child George (case 16). She was a highly-strung, nervous woman, who presented a definite hyperthyroid syndrome - goitre (i.e. swelling of thyroid gland), tachycardia, large pulse pressure, moist skin, fine tremors of hands and eyelids, exophthalmos, and menorrhagia. She did not look anaemic, and presented no symptoms or signs which could not be attributed to the thyroid condition.

She informed me that her mother died six years ago of pernicious anaemia.

I examined the blood as a matter of interest and found the following:-

29.11.36.

Haemoglobin.

86.

R.B.C.

4.620.000.

C.I.

0.93.

Halometer reading.

6.9 u.

Case 43. Mrs D. aet 33.

Mrs D. is a widow and the mother of three children. She is extremely poor, her only source of income being a pension of 17/6 per week. She is of a very neurotic type. She very frequently experiences "heart attacks" in which she describes a praecordial pain of severe degree which does not radiate. During an attack she lies in bed moaning, and describes a feeling of impending death. The blood pressure is always in the region of 120 - 125 systolic, and 70 - 80 diastolic. During an attack she shows a tachycardia - the pulse being in the region of 110 - 120 per minute. Despite exhaustive and repeated examinations, I found no evidence of organic disease, nor could the specialist to whom I sent her. Before affixing an apologetic "functional" label to her case I did a blood count and found the following:-

12.2.37.

Haemoglobin.	91.
R.B.C.	5.030.000.
C.I.	0.91.
Halometer reading.	7.3 u.

Case 44. Helen H. aet 35.

This was a thin miserable-looking patient, who lived in a small one-roomed house with her aged mother. Their combined income (pension and insurance benefit) was 18/- per week. She was unemployed, and had been "on the panel" for eight years. She had a low duodenal/

duodenal ulcer which was diagnosed and demonstrated radiologically ten years ago. On two subsequent occasions the ulcer crater has been demonstrated. Surgical opinion is to the effect that she is unsuitable and unfit for operation. She remains comparatively symptom free by taking alkalis and adhering, as far as her temperament and finances will allow, to a modified Sippy diet. I examined her in November 1936 and found the following.

She was a thin ill-nourished female. Her weight (clothed) was 6 st. 1 lb. and height 5 ft. 4 inches. The pulse rate was 98 per minute, and irregular. There were occasional extra systoles which were made more obvious by exertion. The exercise tolerance test gave a poor response. She was dyspnoeic on exertion. The mucous membranes were pale. There were no tongue or nail changes, and no history of dysphagia was given. The abdomen was poorly clad. There was tenderness but no rigidity in the epigastrium towards the right side of the mid-line. The spleen and liver were not enlarged. A blood examination gave the following result:-

18.11.37.

Haemoglobin.	63.
R.B.C.	4.090.000.
C.I.	0.78.
Halometer reading.	6.9 u.

The Benzidine test was negative on two consecutive occasions./

occasions. ~~Sensidine was positive in one of three~~

I prescribed Ferri. et. ammon. cit. gr. xx, t.i.d. but this had to be discontinued because of severe gastro-intestinal upset. Even smaller doses of scale iron were not tolerated. After cautiously exhibiting saccharated ferrous carbonate and finding it did not upset her I incorporated this preparation into the alkaline powder she was taking - as follows:-

Ferrous carb. sacc.

Calc. carb.

Sod. bicarb.

Mag. carb. pond.

Sig. 3 i, ex aqua t.i.d. p.c.

She was thus getting approximately  $\frac{1}{4}$  of one drachm (gr. xv) of this particular iron preparation thrice daily. She took this powder, which was quite palatable and fairly-well tolerated, for a period of three months. After two months the blood examination showed very little improvement, viz.

14.1.37.

Haemoglobin.	72.
R.B.C.	4.520.000.
C.I.	0.80.

I did not see her again until November 1937.

She had not been taking any iron preparation for six months. The clinical condition was "in statu quo."

The blood examination revealed the following:-

28.10.37.

Haemoglobin.	73.
R.B.C.	4.090.000.
C.I.	0.90.
Halometer reading.	7.2 u.
W.B.C.	7.000.

The/



The Benzidine test was positive in one of three consecutive tests.

With much persuasion she took Ferrous carbonate in increasing doses. In three weeks I had her taking 3 i, t.i.d. of the saccharated preparation. An examination in December gave the following result:-

4.12.37.

Haemoglobin. 82.

There was a definite amount of digestive upset and I finally gave the patient permission to leave off the iron. A subsequent enquiry at the local chemist's revealed the fact that my action had been anticipated by the patient, who had failed to have repeated my original prescription for a iii supply!

She has taken no iron preparation since. The latest figure I have is:-

24.2.38.

Haemoglobin. 74.

I should like to note (and I do not think the matter irrelevant) at this point, the great difference in the matter of exhibition of any particular line of therapy in hospital as opposed to private practice. In the former a few directions are noted on the patients' chart and there the matter ends. In general practice the handing out of a prescription is only the beginning. Information has to be collected from the patient, (the discolouration of the tongue and teeth by scale iron gives the drug an advantage which I did not fully appreciate as a student in hospital clinics) her friends and relations, and the local chemist before any inference can be drawn with regard to the particular therapy adopted. In many cases I have had to invoke the aid of all the parties, and sometimes that of an actual/

actual eye witness. In one particular case I learned that the patient had been taking Bland's pills concurrently with the iron and ammonium citrate I had prescribed - because, as she naively put it 'her friend told her they were good for bloodlessness!'

Case 45. Mrs M.D.

This patient, the mother of two children, was at the time of consultation five months pregnant. She came to make arrangements for her confinement only, and had no complaints. She said she felt as well as she had always done. On examination I noticed that she was very pale and there was a moderate degree of pallor of the mucous membranes. The tongue was pale and atrophic. Direct questioning revealed the fact that there was dysphagia of moderate degree. Vague digestive upset was complained of, which had been present for a period of two years and had become aggravated by the pregnancy. The finger nails were flattened, brittle and cracked. There was no splenic enlargement. The pulse rate was 86 per minute, blood pressure 128 / 78 and the heart sounds were closed and pure. The urine showed no abnormal constituent. The patient's own diagnosis of a five months' pregnancy was confirmed. Clinical examination revealed nothing else of note.

On direct questioning the patient vouchsafed the information/

and the following was noted:-

52.1.30./

information that she had gradually, over a period of six months, become more and more breathless on exertion. She had been vaguely unfit for about six or seven months. She suffered from insomnia and occasional headache. Her husband was in regular employment - despite a marked mitral stenosis - but earned on an average, only about 38/- per week. Mrs D. had no flair for household management. She was not blessed with a full quota of native wit and one can postulate a long-standing minor degree of cerebral anaemia superimposed on a mild degree of original mental deficiency. For three days out of seven the D. family's budget was run on a deficit by the aid of credit from the local tradesmen. The family menu was of the high carbohydrate, low protein, vitamin and iron, and highly astringent, type described in other cases above. Examination of the blood revealed the following:-

21.11.37.

Haemoglobin.	67.
R.B.C.	4.880.000.
C.I.	0.65.
Halometer reading.	6.8 u.
W.B.C.	6.060.

I prescribed Ferrous sulphate gr. iii, t.i.d. and gave her 100 pills. She reported in one month with a specimen of her urine for routine examination. She said she was feeling much more fit. I advised her to have her prescription repeated. At the end of the second month's therapy I arranged a blood examination and the following was noted:-

31.1.38./

31.1.38.

Haemoglobin.	83.
R.B.C.	4.890.000.
C.I.	0.86.
W.B.C.	8.200.

I learned at this point that the patient had not finished her first bottle of pills. To be exact she had only taken 87 pills (four weeks' supply) in approximately two months. I exhorted her to continue her therapy but only saw her once more before her delivery. Her husband brought her sample of urine for routine examination.

15.3.38. Mrs D. was to-day delivered spontaneously of a full time healthy child. She stood the confinement very well. She actually took, in all, 128 pills in the four months following the first blood examination, i.e. six weeks' supply.

Case 46. Mrs T. aet 34.

This patient was a married woman who had borne one child (still born) five years ago. She was a stout, florid woman with rosy cheeks, who presented at first sight the appearance of very good health. She lived alone with her husband, an estate manager, in a comfortable home and knew no economic hardships.

She came complaining of a sore tongue and nothing else. She confessed to slight dyspnoea on exertion and, on direct questioning, to dyspepsia of the hypochlorhydric/



hypochlorhydric type. In describing her symptoms she herself offered the information that she had recently had difficulty in swallowing. The glossitis was her most urgent symptom and she felt certain that she was developing a cancer of that organ. On physical examination it was observed that the tongue was red and angry, the papillae were hypertrophied, and there were crops of minute ulcers along the lateral margins and at the tip. The mucous membranes were suspiciously pale. The finger nails were brittle and cracked, but not flattened or concave. The pulse rate was as follows:-

18.10.36.

Haemoglobin.	75.
R.B.C.	4.400.000.
C.I.	0.85.
Halometer reading.	7.4 u.

She had been employing a great variety of proprietary mouth washes over a period of five to six months. I advised her to discontinue these and pre-scribed Ferri. et. ammon. cit. gr. xx, t.i.d. This she tolerated very well. In two weeks she reported "in statu quo" I prescribed dilute hydrochloric acid to be taken with her meals, and advised her to persist with the iron. After a further two weeks she again reported feeling and looking very much happier. Her symptoms were practically gone and with them the cancerophobia. Unfortunately I have not seen her since.

Case 47. Mrs McD. aet 35.

This patient is a housewife, the mother of two children, who came complaining of severe frontal and occipital headache. Further questioning revealed the fact that she was the subject of menorrhagia, which had been preceded by spells of amenorrhoea. She complained too, of lack of appetite, "indigestion" and "hot flushings" which came on at any time, but especially after taking hot liquid. She had recently been putting on weight. The pulse rate was 78 per minute, and regular. The blood pressure was 170 / 96 but there was no hypertrophy, or, indeed, any demonstrable cardiac lesion. The urinary findings were negative. The retinae appeared healthy. There was no error of refraction. The mucous membranes were pale but no abnormality was detected in the tongue or finger nails. On direct questioning there was elicited a history of dyspnoea on exertion, occasional oedema of the ankles, and a general lassitude and unfitness for house-hold duties, over a period of six months. The early onset of the menopausal syndrome was the tentative diagnosis offered. The blood was examined and the following noted.

21.10.37.

Haemoglobin.	63.
R.B.C.	4.100.000.
C.I.	0.77.
Halometer reading.	6.9 u.
W.B.C.	6.200.

The/

The patient was reassured and, at the same time, advised to lead a restricted life. 'Theominal' and daily morning saline aperients were prescribed. Ferrous sulphate gr. iii, t.i.d. was also exhibited.

One month later she was looking and feeling much improved. The headaches were gone and the patient was generally feeling much more fit.

On 10.12.37. the blood pressure was 150/90. She was much more fit for household duties. On 8.1.38. the blood pressure was 140/92. She had taken approximately 200 ferrous sulphate pills and seemed to be radiating good health. I asked her to discontinue the pills and report in two weeks. The blood pressure was then 142/90 - 20.1.38. The periods were fairly regular and the loss less profuse. The patient was certainly much happier and apparently symptom free. She failed to report and I have no further notes on her case.

Case 48. Mrs D. aet 36.

Mrs D. was the mother of one child. She had consulted my principal two months before I saw her, complaining of lassitude, vertigo, tinnitus, breathlessness on exertion, and oedema of the ankles. He prescribed Ferrous sulphate gr. iii, t.i.d. which she had been taking regularly for the previous two months.

On examination, apart from a moderate pallor of the/

the mucous membranes no abnormality was detected.

Her husband earns on an average 35/- per week. She, her husband, and child apparently subsist quite well on this income and think nothing of it. The blood findings were:-

7.11.37.

Haemoglobin	89.
R.B.C.	4.690.000.
C.I.	0.96.
W.B.C.	6.000.
Halometer reading.	7.4 u.

Case 49. Mrs C. aet 36.

This was a very pale-faced, grossly over-weight woman. She had three children, and was breast feeding the youngest aged two months. She had a dull, weary, pathetic expression. Her husband was unemployed and the income was less than 25/- per week for this family of five. She complained of extreme lassitude, breathlessness and palpitation on exertion, disinclination for food, and said she was "completely run-down."

On examination there was marked pallor of the mucous membranes. She had four carious teeth, but the tongue was normal although pale. The nails were flattened and four of them manifested a definite concave spoon-shape. The pulse rate was 94 per minute, but no other cardiac abnormality was noted. There was oedema of the face and ankles. The urinary findings were negative. Cerebration was definitely retarded/



retarded and the patient was mentally dull, inattentive, and obtuse. The blood findings were as noted:-

11.11.36.

Haemoglobin.	60.
R.B.C.	3.940.000.
C.I.	0.76.
Halometer reading.	7.0 u.

Ferrous sulphate gr. iii, t.i.d. was prescribed and there was a most gratifying and dramatic response after only two weeks therapy. Fortunately the patient's husband obtained employment shortly afterwards, but rather unfortunately for me she left the district and I have no more notes on the case.

Case 50. W.C. aet 38.

This patient was an oxy-acetylene burner at the local shipbreaking yard. He was not my patient, but I met him in consultation when he reported for periodic examination at the shipbreaking yard. There are some 40 to 50 burners examined every month and this is the only case I have had reported to me by my principal (who holds the appointment of examiner) in the course of two years.

The patient complained of vague colicky abdominal pains and severe, obstinate constipation. He felt vaguely "out of sorts" but had no other definite complaint. On examination it was noted that he was a pale-faced man of excellent physique. There was moderate/

moderate pallor of the mucous membranes. No blue line was observed on the gums, and there were three carious teeth. There was a very slight weakness of the extensor tendons of both wrists - not amounting to wrist drop - but simply a lessened resistance to forcible flexion. The blood pressure was not raised - 132/80 - and the urinary findings were negative. The blood picture was as follows:-

3.12.36.

Haemoglobin.	81.
R.B.C.	4.070.000.
C.I.	1.01.
Halometer reading.	7.6 u.

The film showed up well with Leishman's stain. The cells were fairly uniform in size and well filled with haemoglobin. A very few poikilocytes were observed. No nucleated red cells were seen but there was a definite punctate basophilia which I estimated at 4%. The patient was given another job in the yard and not allowed to use the 'burners' for one month. Symptomatic treatment was carried out by his own doctor and in four weeks he returned to his job apparently in good health.

Case 51. Mrs P. aet 39.

This woman is the mother of one child aged 10 years and the wife of a general practitioner. Herself a graduate of medicine she found it difficult to refrain from giving me a list of diagnosed conditions instead of/

of an impartial history of symptoms and signs.

When a student she had an attack of influenza of unduly prolonged duration, in which the physical signs in the chest predominated. In 1922 she had a frank haemoptysis and pulmonary tuberculosis was diagnosed. She was sent to Switzerland and thence to South Africa where she made a speedy and apparently complete, recovery. She returned to take up the rather strenuous occupation of 'wife to a busy general practitioner' at the same time producing and bringing up a daughter. For the past five years she had complained of vague abdominal discomfort and lack of energy, an increasing degree of tachycardia, and breathlessness on exertion. For the preceding two months she had attacks of paroxysmal tachycardia or frequently recurring extra systoles. They were accompanied by a feeling of acute apprehension and a sense of impending death. A consultant saw her in August 1936 and diagnosed visceroptosis associated with a vasomotor upset which was largely functional in origin. Suggestive treatment was adopted and bromides exhibited. The patient did not improve and even with the help of bromides suffered from severe insomnia, which was associated with an exasperating degree of fatigue and weariness. I examined her in October 1936 and found the following.

She was a small sparely-built woman, five feet two inches in height, and weighing 6 st. 10 lbs. The face was very pale and the mucous membranes were moderately pale. There were no tongue or nail changes.

The/

The pulse rate was 98 per minute and frequent extra systoles were brought on by even moderate exertion. The blood pressure was 106/70. I could not detect the presence of any organic disease of the heart or lungs. The blood picture was:-

16.10.37.

Haemoglobin.	70.
R.B.C.	4.530.000.
C.I.	0.78.
Halometer reading.	6.8 u.
W.B.C.	7.400.

The following day the menstrual period began and the loss was very profuse. The patient was prostrated and confined to bed. The blood pressure fell to 92/60 and there were frequent alarming attacks of paroxysmal tachycardia. They would come on suddenly and pass off equally suddenly. They lasted from a few seconds, up to 2 - 3 minutes, during which the pulse was too fast to be counted. These attacks could only be controlled by firm pressure over the carotid sinus in the neck. Expectant treatment was adopted. When she had settled the blood examination was repeated and the following noted:-

13.11.36.

Haemoglobin.	61.
R.B.C.	4.100.000.
C.I.	0.74.
Halometer reading.	6.9 u.

Iron was exhibited cautiously - one, two, then three tablets daily of Ferrous sulphate gr. iiii, t.i.d. After she had taken 100 tablets the blood was examined and/



and the following noted:-

14.12.36.

Haemoglobin.	90.
R.B.C.	4.820.000.
C.I.	0.94.
Halometer reading.	7.2 u.

The patient was enormously improved and was naturally impressed by the fact that a definite cause for her so called neurotic complaints had been demonstrated. She continued in good health throughout the winter. She had previously had recurrent attacks of pharyngitis and tonsillitis, which took a great deal out of her, and frequently caused the onset of extra systoles and tachycardia. Her pulse was now in the region of 80.

I examined her one year later. She appeared to be in perfect health. She said she could 'feel' when she required a course of iron, and had of her own accord resorted to it in time of need. When she could "feel her haemoglobin level dropping" the regular taking of ferrous sulphate rapidly brought about an improvement.

Case 52. Miss J. N. aet 39.

This patient is a shop assistant at the local chemist's. She is a sister of Miss N. case 24. She stated that for the past ten years she had been the subject of bilious attacks (headaches, nausea and vomiting) which are undoubtedly allergic in nature. They/

They can be induced by the taking of syrup, honey, treacle, or oranges, and by the inhalation of pollen dust. She says she has always been anaemic and has taken the following prescription for nine months out twelve for the past ten years. She finds that it is, in her case, a specific against biliousness.

Liq. Ferri. perchlor.  
 Liq. Ammon. acet. conc. 1/7 aa M. xv.  
 Tr. nucis. vom. M. iii.  
 Syrup simplex.  
 Glycerini. aa M. xx.  
 Aqua. chlorof. ad.

Sig. ex aqua. t.i.d. p.c.

She is frequently 'run-down,' has swollen ankles, and is easily tired, but the above 'tonic' causes a very rapid disappearance of these symptoms.

I examined her in February 1937 during one of these spells. She said she was "fagged out" and felt unfit for her rather arduous duties. On examination a definite pallor of the mucous membranes was noted. There were no tongue or nail changes. There was a slight degree of oedema of the ankles. The urinary findings were negative. The pulse rate was 80 per minute and no other abnormality was detected. The blood was examined and the following noted:-

21.2.37.

Haemoglobin.	78.
R.B.C.	4.640.000.
C.I.	0.85.
Halometer reading.	7.0 u.

I prescribed Ferrous sulphate gr. iiii, t.i.d.

and/

and after two weeks the following was noted:-

7.3.37.

Haemoglobin.	86.
R.B.C.	4.800.000.
C.I.	0.89.
Halometer reading.	7.2 u.

She continued taking the pills and after two more weeks the following was noted:-

29.3.37.

Haemoglobin.	112.
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The patient was very much improved in her general health, but she confessed that she preferred her old prescription, as she felt she could rely upon it as a specific against biliousness.

Case 53. John D. aet 39.

This patient consulted me on account of praecordial pain. He said he had become gradually more breathless for the past two years. He had lately, been feeling "out of sorts." He had recently noticed that he got a "pain at his heart" when he climbed stairs too quickly. He is a shop keeper earning a fair salary and partakes of a mixed generous diet.

On examination it was noted that he was very pale, the conjunctivae and lips being especially so. He had no oedema. His pulse was regular ( 76 per minute ) but of poor volume. His apex beat was normal in character and position. His blood pressure was 120/88.

It/

It was discovered on further enquiry that he had a moderate degree of oligouria. On four successive but days he passed 28, 30, 29 and 32 ounces of urine in the 24 hours. In none of these specimens could I find a trace of albumin. I had no facilities for preparing a centrifuged specimen. I examined the blood and found the following:-

23.1.38.

Haemoglobin.	101.
R.B.C.	5.370.000.
C.I.	0.95.
Halometer reading.	7.3 u.
W.B.C.	7.400.

He was referred to a specialist who could find no evidence of organic disease. X-rays and electro cardiographs were negative. It was discovered that the patient was heavily in debt and had many financial worries. He responded well to psychotherapy, and the exhibition of bromides. The pallor of the face and mucous membranes, however, was not decreased.

Case 54. Mrs H. aet 40.

This lady is the mother of three children and the wife of a dentist. She was a state registered nurse and although she appreciates the general principle of dietetics, her meals were often eaten in a haphazard manner, fitted in at any time when domestic exigencies would allow. She said she was 'fairly fit' but confessed that she had probably become more easily tired over/



over an approximate period of one year. She looked very pale, and the mucous membranes were blanched, but this she dismissed as her usual state. Apart from the marked pallor and history of being vaguely unfit and easily tired, I could detect nothing else of note in her history or from clinical examination. The blood picture was as follows:-

3.11.37.

Haemoglobin.	71.
R.B.C.	4.290.000.
C.I.	0.85.
W.B.C.	6.400.
Halometer reading.	7.2 u.

I prescribed Ferrous sulphate gr. iii, t.i.d. and in two weeks she was noticeably much improved. She was eating more and enjoying her food. She felt very much more fit for her household duties. After taking 100 tablets she showed the following:-

7.12.37.

Haemoglobin.	105.
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Case 55. Mrs M. aet 41.

This patient is the mother of one child aged 14. Her husband earns 40/- per week. She lives in a small, dark, ill-ventilated house, and goes out very little. Her dietary arrangements are very inadequate. Fried, greasy dishes, and very little protein, vegetable, fruit, and iron-rich foodstuffs, are its chief features. Mrs M. consulted me on account of oedema of the ankles. She/

She said this had lasted in varying degrees for three years. She had been very 'run-down,' was easily tired, and had had palpitation on exertion, for a period of one year. She had no appetite, complained of marked dysphagia, and a feeling of weight in the epigastrium after taking a meal; she had occasional bouts of severe diarrhoea. On examination it was noted that she was a stout pale-faced woman. The mucous membranes looked ex sanguine. There was slight puffiness of the face and marked oedema of the ankles. The pulse rate was 92 per minute. The heart sounds were closed and pure. There was a slight trace of albumin in the urine. The tongue was smooth and atrophic; the finger nails were cracked - she was the most severe case of this I have seen. Two of the nails showed a definite tendency to concavity. Nothing else was noted on physical examination. The patient said she had always been anaemic, and in her early teens had been very ill with 'bloodlessness.' (Chlorosis ?).

The blood was examined and the following noted.

10.2.37.

Haemoglobin.	50.
R.B.C.	3.330.000.
C.I.	0.76.
Halometer reading.	6.5 u.
W.B.C.	8.200.

The film showed a marked microcytosis and 'ring forms' were numerous. Although some cases had a lower colour index she is the most marked case of microcytosis I have seen.

I/

I prescribed Ferrous sulphate gr. iii, t.i.d. She said she could not swallow these. She crushed up the tablets and took them in her tea! The blood was again examined.

14.3.37.

Haemoglobin.

75.

I found that she had only taken approximately half, of 100 prescribed tablets. She said she felt much better. The oedema had disappeared with rest. The pulse rate was more steady, and the patient was in much better spirits. There was no albuminuria. She failed to report and on enquiry at the local chemist's it was revealed that she did not have her prescription repeated.

Case 56. Mrs S. aet 41.

This patient is the mother of one child, aged 7 months. She stated that she had been in vague ill-health since the birth of her child and had recently been "very short of breath." The actual confinement was apparently normal (I did not attend her) but her friends noticed that she had been very pale since about the time of her confinement. On examination it was noted that she was a tall, pale-faced woman. The mucous membranes were blanched. There was a slight degree of oedema of the face and ankles. She had no albumin/

albumin in three consecutive specimens of urine. The blood pressure was 120/70 and the heart sounds were closed and pure. The pulse rate was 92 per minute at rest. The exercise tolerance test gave a very poor result. There was breathlessness on even moderate exertion. I discovered nothing else of note on physical examination.

She had been taking Ferri. et. ammon. cit. gr. xv, t.i.d. for four months before I saw her. I examined the blood and the following was noted:-

30.10.36.

Haemoglobin.	45.
R.B.C.	4.170.000.
C.I.	0.54.
Halometer reading.	6.8 u.
W.B.C.	8.600.

I prescribed Liq. Ferri. perchlor. M xv, t.i.d. and saw her one month later. She was not improved and the following was noted:-

6.12.36.

Haemoglobin.	44.
R.B.C.	3.930.000.
C.I.	0.56.

She was given a prescription for Ferri. et. ammon. cit. gr. xxx, t.i.d. but she could not tolerate this dose. It was reduced to gr. xx, t.i.d. After two weeks she said she was feeling better but failed to report for a blood examination.

I met her fortuitously three months later when the blood condition was:-

8.3.37/



8.3.37.

Haemoglobin.

56.

I prescribed Ferrous carb. sacch. 3 i, t.i.d. but did not see her again for six months. I had been attending her mother and induced the patient to come for a blood test. The following was noted:-

27.10.37.

Haemoglobin.

45.

R.B.C.

4.390.000.

C.I.

0.52.

Halometer reading.

7.1 u.

W.B.C.

7.400.

I heard that she had taken no iron in the interval. The periods had been scanty and her general condition was 'in statu quo' but evidently not sufficiently distressing to cause her to do anything about it. She was quite contented to 'muddle along.' I pre-:scribed Ferrous sulphate gr. iiii, t.i.d. She took approximately 80 of these pills and was greatly im-:proved. I did not see her again for three months. I was called in late one night. The patient had profuse vaginal blood loss. She had not had a period for 9 weeks and suspected a miscarriage. There was no other evidence of this, however; the bleeding settled, and stopped entirely in ten days when her blood count was shown to be:-

9.2.38.

Haemoglobin.

67.

R.B.C.

4.660.000.

C.I.

0.73.

I insisted in strong terms that she continue taking/

taking Ferrous sulphate gr. iii tablets regularly, four tablets daily. After three weeks she was very much improved and the following noted:-

3.3.38.

Haemoglobin.

86.

Case 57. Mrs D. aet 41.

This lady is the mother of four children, the youngest being 10 years old. She came complaining of weakness, tinnitus, dizziness, palpitation on exertion, and disinclination for food. She was a stout, florid woman, but the mucous membranes were pale. She said she had gradually become more unfit over a period of six months.

On examination pallor of the mucous membranes was noted in contrast to the injected blood vessels of the malar region. The tongue was pale but apparently normal. The finger nails were flattened and a slight degree of koilonychia was present. The pulse rate was 86 per minute and the rhythm regular. There was a soft systolic murmur at the pulmonary area. The blood pressure was 130/82. There were no other positive findings at examination. The blood picture was found to be:-

4.11.36.

Haemoglobin.

70.

R.B.C.

4.620.000.

C.I.

0.76.

Halometer reading.

7.0 u.

W.B.C.

6.200.

Ferri./

Ferri. et ammon. cit. gr. xxx, t.i.d. was prescribed. It caused mild digestive upset. The bowels, which had hitherto been rather costive, were definitely loose. She persisted with the scale iron for four weeks and improved in a most dramatic and gratifying manner. I did not see her again till I was called in to treat her daughter for quincy. Mrs D. was persuaded to have a blood test done. She said she was feeling 'quite well' and had no complaints. She said she often felt tired in the evening, but put that down as 'normal.' The blood picture was very similar to that noted one year previously. viz.

5.11.37.

Haemoglobin.	75.
R.B.C.	4.410.000.
C.I.	0.85.
Halometer reading.	7.1 u.

Ferri. et. ammon. cit. gr. xxx, t.i.d. was prescribed.

Case 58. Mrs H. aet 42.

This was a married, nulliparous woman. She was employed in the kitchen of a nearby hospital. She came to see me, on her day off, complaining of dullness, lassitude, and insomnia. She felt in need of a tonic. She complained of nothing else. On examination a marked pallor of the mucous membranes was noted. She had no nail changes. The tongue appeared healthy, but in response to direct questioning she said it had been sore/

sore for some time. Nothing else was noted at clinical examination. The blood picture was as follows

1.12.36.

Haemoglobin.	65.
R.B.C.	3.850.000.
C.I.	0.84.
Halometer reading.	6.8 u.

Ferri. et. ammon. cit. gr. xxv, t.i.d. was prescribed, and she made a rapid improvement. After three weeks her haemoglobin was estimated:-

22.12.36.

Haemoglobin.	83.
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She continued taking the iron preparation fairly regularly (I checked her prescription at the local chemist's) and the blood was examined after three more weeks:-

18.1.37.

Haemoglobin.	92.
--------------	-----

I asked her to report in one month's time. She did so and was looking and feeling in perfect health. The blood was examined and the following noted:-

16.2.37.

Haemoglobin.	101.
R.B.C.	5.300.000.
C.I.	0.95.

Case 59. John R. aet 42.

This man was the subject of a moderate degree of disseminated sclerosis of four years' duration. He complained of heaviness and weakness and stiffness in the lower limbs. He also had numbness and paraesthesia of/



of both upper and lower limbs. The deep reflexes were exaggerated. He had a thick, slurring speech; the tongue showed a fine tremor; there was a coarse tremor of the hands and fingers made worse by voluntary movement; and the patient had been the subject of recurring attacks of diplopia. The acts of micturition and defaecation had not been interfered with. He had no other complaints. The blood pressure was 142/84.

Although there was little doubt about the nature of the diagnosis, I examined the blood to rule out the possibility of sub-acute combined degeneration of the cord, associated with latent pernicious anaemia.

The blood picture was:-

18.10.36.

Haemoglobin.	103.
R.B.C.	5.210.000.
C.I.	0.99.
Halometer reading.	7.2 u.

Case 60. Mrs S. aet 42.

This patient was a very obese pale faced woman, who had borne seven children. Her husband is a labourer earning on an average 35/- to 40/- per week, and she has, for the greater part of her married life, been engaged in a grim struggle for existence. The family resources have, however, been increasing for the past six years as each of her children becomes old enough to obtain employment at the local papermill. Mrs S.' complaints were many and various. The chief of/

of these were dyspnoea and palpitation on exertion, which had become much worse in the past six months. She felt quite 'worn out' and unfit for work. The least exertion was a trouble to her. She had suffered from menorrhagia for the past year and had had prolapsed haemorrhoids, which, from time to time, would bleed profusely, for the last six years. In answer to a leading question she said she had had dysphagia for about six months. On examination it was observed that she was a pale-faced female, grossly overweight. The pulse rate was 88 per minute at rest, and the blood pressure 120/72. There were systolic murmurs at the mitral and pulmonary areas, and over the great vessels in the neck. The cardiac rhythm was, however, regular. There was slight oedema of the face and ankles and the urine showed a trace of albumin. The finger nails showed a well marked koilonychia. The blood was examined and the following noted:-

28.10.36.

Haemoglobin.	42.
R.B.C.	3.920.000.
C.I.	0.54.
Halometer reading.	0.6 u.
W.B.C.	6.520.

I prescribed Ferrous sulphate gr. iii, t.i.d. and she managed to swallow these quite well despite the dysphagia. She made a very rapid and gratifying improvement and was most enthusiastic about her treatment. Her appetite was markedly improved. She was much less breathless and when I saw her one month later there/

there were no cardiac murmurs. The dysphagia had gone and the patient was looking much happier. The menorrhagia had settled, the menstrual loss being considerably less at the next period. The blood picture was much improved. viz.

7.12.36.

Haemoglobin.	87.
R.B.C.	5.430.000.
C.I.	0.81.
Halometer reading.	6.8 u.

I urged her to continue with the therapy but she failed to report.

I saw her about one year later when she said she was again needing a 'tonic' and as she had lost her prescription would I "give her a line to get some more pills." I gave her a prescription for Ferrous sulphate gr.iii, and asked her to report for a blood count. She did so three weeks later and the following was noted:-

22.11.37.

Haemoglobin.	72.
R.B.C.	5.350.000.
C.I.	0.68.
W.B.C.	7.200.
Halometer reading.	6.6 u.

She said she felt very much better than she had been before commencing the pills three weeks previously. I urged her to continue taking the pills but she failed to report again.

Case 61. Mrs. C. aet 43.

This patient is the wife of one of the local general/

general practitioners and has borne two children, and had two miscarriages. There was definite pallor of the face but the mucous membranes were not pale. Indeed, she presented no signs of anaemia and was looking and feeling perfectly fit. She is a sister of Mrs P. case 51.

The blood examination was as follows:-

1.11.36.

Haemoglobin.	102.
R.B.C.	4.950.000.
C.I.	1.03.
Halometer reading.	7.2 u.

Case 62. Mrs F. aet 45.

This patient had two months previously had the menopause induced by the exhibition of deep X-rays. The indication for this had been profuse menorrhagia, and vasomotor disturbances. She had no symptoms and appeared to be in good health. I examined her blood as a matter of interest to see if there was any marked anaemia resulting from this spell of one year's menorrhagia. The following was noted:-

22.11.36.

Haemoglobin.	98.
R.B.C.	4.820.000.
C.I.	1.02.
Halometer reading.	7.3 u.

Case 63. Mrs C. aet 45.

This lady, the mother of one child, had not yet passed /



passed the menopause. She was caretaker at a golf club. She was of a highly-strung, emotional type, who gave me a history of vague abdominal discomfort, referred to the epigastric region, sometimes amounting to pain, and indefinitely related to meals. The appetite was poor. She was greatly troubled with flatulence and pain after eating fat or greasy meals. She had a sore tongue. She was troubled with insomnia and headaches she said. One was inclined, at least after the second or third consultation, to postulate a functional origin for a great many of her complaints. The approaching menopause may have been, in a measure, responsible, but there was an underlying anaemic factor which will be evidenced below.

Examination revealed a moderate degree of pallor of the mucous membranes. The tongue was clean and apparently healthy. The nails had a normal conformity. There was tenderness on pressure over the epigastrium and pain on deep palpation over the region of the 1Xth costal cartilage when the patient took a deep breath. The benzidine test was negative and the urine showed no abnormal constituent. The blood pressure was 140/82. The blood was examined and the following noted.

19.10.37.

Haemoglobin.	78.
R.B.C.	4.680.000.
C.I.	0.85.
Halometer reading.	7.1 u.
W.B.C.	7.400.

I/

I prescribed Ferrous sulphate gr. iii, t.i.d. and saw her two weeks later. She had found great difficulty in taking the pills as they occasioned rather severe gastro-intestinal upset. I examined the blood one month later - she had only taken 58, instead of 100 pills - and the following was noted:-

25.11.37.

Haemoglobin. 89.

I stopped the iron therapy and prescribed dilute hydrochloric acid with meals and a gentian and strychnine mixture before meals. viz.

Acid hydrochlor. dil.	M x.
Liq. strych. hydrochlor.	M iii.
Glyc. pepsini.	M xx.
Inf. gent. co. ad.	3 ii.
Sig. 3 ii ex. aqua. t.i.d. a/c.	

This merely aggravated the gastric distress and the pain in the region of the gall bladder became more acute. She was referred to hospital where a thorough examination failed to reveal any radiological evidence of disease of the biliary or renal tracts. There was no radiological evidence of peptic ulcer. A test meal was not done. I prescribed alkalis with a modified Sippy diet, and this afforded considerable relief of the alimentary symptoms. Ferrous sulphate was again cautiously exhibited and after two weeks the patient was considerably improved. The appetite was good and the vague complaints of lassitude, weariness, and insomnia were very much less urgent.

Case 64/

Case 64. Mrs McC. aet 46.

This patient is the mother of three children. She came complaining of being run-down, easily tired, and 'rather out of sorts.' She felt unfit for her work, and requested a tonic. She had no other complaints. She had not yet passed the menopause. On examination it was noted that she was an obese, pale-faced woman. The mucous membranes were moderately pale. The tongue was atrophic. The pulse rate was 80 per minute. The blood pressure was 132/78 and the heart sounds closed and pure. Nothing else was noted in the clinical examination. A blood count was done and the following noted:-

11.2.37.

Haemoglobin.	78.
R.B.C.	4.410.000.
C.I.	0.89.
Halometer reading.	7.3 u.
W.B.C.	8.200.

I prescribed Ferri. et. ammon. cit. gr. xxx, t.i. d. which she took regularly, and I examined the blood after four weeks. The following was noted:-

14.3.37.

Haemoglobin.	110.
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The patient was in radiant spirits and was feeling very fit indeed. I have not seen her since.

Case 65. Mrs Annie S. aet 47.

This patient is the mother of three children.

The/

The father is unemployed. She had, unfortunately, received treatment (at the hands of my principle) for six weeks before I examined her. She said she was feeling enormously improved since she had started taking the pills (ferrous sulphate gr. iii) but was not yet quite fit. Her chief complaint had been breathlessness and lack of energy. She could not walk up a hill. She said she was very nervous and "jumpy." She suffered from severe insomnia. Her ankles had been swollen and she had had palpitations. Her periods had been very profuse and frequently occurring for the past year.

On examination it was noted that she was a pale-faced woman, inclined to adiposity. The mucous membranes were very pale. The tongue was apparently normal but there was considerable oral sepsis from carious teeth and associated pyorrhoea alveolaris. The finger nails were brittle and cracked but not concave. There was no history of dysphagia. The pulse rate was 82 per minute and the blood pressure 145/90. The heart sounds were closed and pure. The urine showed no abnormal constituent. There was no oedema or any other positive finding. The blood picture was as follows:-

16.10.37.

Haemoglobin.  
R.B.C.  
C.I.  
W.B.C.

67.  
4.160.000.  
0.81.  
6.600.

I/



I advised her to continue taking the pills, but she failed to report again.

Case 66. Mrs D. aet 47.

This patient, who had one child, came to consult me on account of menorrhagia. She said the loss was taking a great deal out of her and that she had for the past seven or eight months become increasingly listless and easily tired. She had frequent headaches and hot flushings of the face. She felt she was unfit for her routine household duties. Her appetite was very poor and she suffered from indigestion of the hypo-secretory type. The bowels were obstinately constipated. She had no glossitis or dysphagia, and presented no other symptoms, either spontaneously or in answer to leading questions. On examination it was observed that she was of rather florid visage and was of average height and weight. The mucous membranes were not clinically pale. The pulse was regular and of good volume. The blood pressure was 146/98. Nothing else was noted. The blood picture was:-

1.11.36.

Haemoglobin.	69.
R.B.C.	4.930.000.
C.I.	0.70.
Halometer reading.	7.2 u.
W.B.C.	8.300.

I prescribed Ferrous sulphate gr. iiii, t.i.d. and in one month she was considerably improved. I did not

see/

see her again till one year later. I discovered that she had taken the pills for two months. She had not had a period since January 1937 and presumably the menopause has become established. She appeared to be in perfect health and the blood figures were:-

5.2.38.

Haemoglobin.	96.
R.B.C.	5.420.000.
C.I.	0.89.

Case 67. Mrs M. aet 48.

This patient was the mother of one child. She had developed a post menopausal bleeding two years ago which was traced to a carcinoma of the cervix uteri. The condition was treated with radium locally and she had a course of deep X-ray therapy carried out later. She was advised to keep in touch with her doctor and to consult him at regular intervals. At the time of examination there was no evidence of local mischief. The patient said she was in fairly good health. She complained, however, of a certain degree of lassitude, anorexia and insomnia. She had no other complaints. She had a wan, weary expression. The complexion had a sallow, pasty appearance, rather reminiscent of that of some cases I have seen of early malignant cachexia. She was of normal weight and height, but she said she had previously been overweight and had lost  $1\frac{1}{2}$  stones in the past two years. The mucous membranes were pale but/

18.10.38./

but nothing else was made out on physical examination.  
The blood picture was:-

28.11.37.

Haemoglobin.	85.
R.B.C.	4.020.000.
C.I.	1.06.
Halometer reading.	7.4 u.
W.B.C.	8.500.

The blood film showed nothing of note. The cells were rather large and fairly uniform in size and shape, and well filled with haemoglobin. It was thought that there was a toxic effect upon the bone marrow, the result of the malignancy or the radium emanations, or possibly both. She was given campolon, 4 c.c. weekly, and in one month seemed to be improved, but no further blood examination has, up to date, been performed.

Case 68. Davina S. aet 48.

This patient was a nulliparous woman. She was a domestic servant. Her complaints were dullness, lassitude, anorexia, dizziness, and severe constipation. She had been treated for two months before I saw her, and had been taking regularly Ferri. et. ammon. cit. gr. xv, t.i.d. She said she was very much the better of her tonic. At examination there was very little to be made out. She was a highly strung, nervous woman, approaching the menopause. The mucous membranes were pale, but only slightly so. She had many carious teeth. There were no tongue or nail changes. Nothing else was noted. A blood count revealed the following:-

18.10.36./

18.10.36.

Haemoglobin.	78.
R.B.C.	5.020.000.
C.I.	0.78.
Halometer reading.	7.2 u.

I increased the dose of scale iron to gr. xxx, t.i.d. and she tolerated this dose well. She said she felt much better, but failed to report again.

Case 69. Mrs McI. aet 52.

This patient had passed the menopause five years ago. She is the mother of four children. Her husband is a manual labourer. She came complaining of headaches which were shown to be due to eyestrain. She had no other complaints. In answer to a leading question she said she had been frequently 'bloodless and run down' in her youth. Physical examination yielded entirely negative results. I decided to examine her blood as a control. The following was noted:-

31.10.37.

Haemoglobin.	96.
R.B.C.	4.780.000.
C.I.	1.01.
Halometer reading.	7.2 u.
W.B.C.	7.200.

Case 70. William B. aet 52.

This man was a farmer. He was of the sturdy pyknic type, and had a ruddy complexion. He consulted me about an attack of "indigestion." He had for more than/



than twenty years been the subject of these attacks, which were characterised by pain in the epigastrium, coming on an hour after meals and occasionally waking him up in the night. He frequently had heartburn and waterbrash, and these were always relieved by the taking of baking soda, or some other alkali. He had been in the habit of getting a prescription for alkali powder at intervals, from his doctor, for a period of seven or eight years. When I saw him he had no complaints other than those attributable to hyperchlorhydria, with a possible associated peptic ulcer.

He had a florid complexion and the mucous membranes were not pale. The stools showed the presence of occult blood. Nothing else was made out by physical examination, and a blood count, performed as a matter of interest, showed the following:-

18.10.36.

Haemoglobin.	86.
R.B.C.	5.950.000.
C.I.	0.72.
Halometer reading.	7.1 u.

Case 71. Mrs M. aet 53.

This patient is the mother of seven children, one of whom is Mrs A. (case 41). She came complaining of breathlessness on exertion, which had gradually become worse, over a period, she thought, of one year. Her last menstrual period had occurred four months previously/

previously. She said she was absolutely exhausted, and that the slightest exertion caused the onset of "palpitations" of a most alarming nature. She had never had a good appetite, she said, but it had lately been even more impaired. Her ankles were often swollen after a hard day's work. She complained of "hot flushings" and the usual vasomotor disturbances associated with the menopause.

On examination it was observed that she was a very stout, heavily built woman. The face was not noticeably pale - due to the presence of a mild degree of Rosaceae. There was definite pallor of the mucous membranes. The tongue was sore but appeared normal. The nails were short, with irregular cracked edges, and flattened. The pulse rate was 104 and the rhythm was regular. There was a soft blowing murmur at the mitral area. A slight degree of oedema of the ankles was present and the urine showed a slight trace of albumin. The blood pressure was 125/70.

No other findings were noted at examination and a blood count revealed the following:-

28.9.37.

Haemoglobin.	52.
R.B.C.	4.260.000.
C.I.	0.61.
Halometer reading.	6.6 u.
W.B.C.	7.500.

Ferrous sulphate gr. iii, t.i.d. was prescribed, and after two weeks the patient was greatly improved.

I/

I saw her at the end of one month, when she had taken 100 pills. She was exceedingly grateful for her sudden and gratifying improvement and was amazed that the pills could have done so much good. (She belonged to that surprisingly common group of patients who think that a doctor's sole function is to prescribe medicine in the form of a bottle!) The patient's 'palpitation' and dyspnoea were considerably relieved. Her appetite had increased; there was no oedema of the ankles or face and she was feeling much more fit. There were no cardiac murmurs. The blood level was:-

3.11.37.

Haemoglobin. 81.

I asked her to continue taking the pills for one more month, and when she reported after taking her second 100, the following was noted:-

6.12.37.

Haemoglobin. 110.

There had not been a further menstruation and it was presumed that the menopause had become established.

Case 72. Mrs L. aet 56.

This patient is the mother of Annie L. case 20. I met her fortuitously while treating the daughter and the following was noted.

She is the mother of two children. She had passed the menopause four years ago. She said she felt/

felt vaguely 'out of sorts,' was easily tired, and suffered from flatulent dyspepsia. Examination revealed that she was a rather pale-faced woman, inclined to adiposity. The mucous membranes were definitely pale. The pulse was regular and there was no tachycardia. The blood pressure was slightly lowered 122/68. Nothing else was noted at the time of examination.

The blood picture was as follows:-

24.11.36.

Haemoglobin.	85.
R.B.C.	4.590.000.
C.I.	0.94.
Halometer reading.	7.1 u.

This patient had not consulted me of her own accord. I had simply arranged to examine her blood as a control against that of her daughter. I prescribed Ferrous sulphate gr. iii, t.i.d. but I have not seen her since.

Case 73. Mrs R. aet 57.

Mrs R. is the mother of Georgina, case 21. She has borne three children, and passed the menopause eight years ago. In November 1936 she had an attack of acute cystitis, and I took the opportunity then to examine her blood. She was a pale faced woman, inclined, like so many of her age and class, towards adiposity. The mucous membranes were moderately pale. There/



There were no tongue or nail changes, or, indeed, anything else to be made out except the cystitis, referred to above, which had been caused and aggravated by a marked degree of prolapsus uteri. The blood picture was as follows:-

29.10.36.

Haemoglobin.	104.
R.B.C.	5.440.000.
C.I.	0.96.
Halometer reading.	7.2 u.

Case 74. George H. aet 58.

This case was an extremely interesting one, in that it was a case of severe anaemia occurring in a middle-aged male.

The patient stated that he enjoyed moderately good health up till about four years ago. He then had vague digestive upset and a feeling of distension in the epigastrium - he said his stomach was swollen. He had a great many carious teeth. Three years ago his doctor advised him to have all his teeth extracted with a view to improving his general health, and his gastric condition in particular. When this had been done (three years ago) he had suffered from an alarming haemorrhage immediately afterwards. This was controlled and gradually ceased, but only after vigorous local treatment, which was carried out for three or four hours. He said he was confined to bed for two weeks/

weeks after this as he was so weak, from loss of blood. He said he had had a similar bleeding eight years ago, which, although not so severe, had caused considerable 'weakness.' The source in this case was haemorrhoids of severe degree. He had suffered from bleeding haemorrhoids irregularly for a period of ten years. He did not think he bled unduly when he cut himself, and there was no available evidence of any haemophilic taint in the family stock. He owned, and, with the help of his sons, managed, a small farm. He was extremely abstemious as regards his eating habits, and in spite of the resources available to him, ate no fruit or fresh vegetables. I enquired specifically about this point, as he had a peculiarly dry, rough, skin, which taken in conjunction with the findings stated below, rather suggested an element of Vitamin C. deficiency. As regards his other habits, he smoked about 3 ounces of tobacco per week, and consumed at least one pint of beer daily.

At the time of examination, the patient, who was still helping to manage his farm, complained of the following - extreme weakness and lassitude of one year's duration; weariness, and a tendency to fall asleep at any time; swelling of the ankles towards the end of each day; dyspnoea on moderate exertion, such as walking up a gentle slope; numbness and paraesthesia of both upper limbs - and to a lesser degree, of the lower limbs; /

limbs; vague pain in the region of the shoulder and knee joints; lack of appetite amounting to a dislike for food; and a "bad taste" in his mouth. He was of rather small stature, but was not emaciated. He was mentally lethargic and dull. His general intelligence was below par.

On examination it was noted that he was a very pale-faced man who looked ill. Superimposed on the deathly pallor of the face was a lemon yellow tinge. The sclera were not jaundiced but there was a large ecchymosis under the conjunctiva of the right eye. Petechial haemorrhages were observed subcutaneously over the body. These were most frequent on both fore-arms, the right arm, and, to a lesser extent, scattered over both legs. They were absent from the trunk. The mucous membranes were blanched. The breath was foul smelling. The tongue was irregularly coated with a thick, brownish-yellow fur, and did not appear to be atrophic. There was no oedema of the limbs, but a slight degree of puffiness of the face was noted, and the eyes were puffed. The pulse rate was 86 and the blood pressure was low, 120/66. There was a slight degree of cardiac dilatation evidenced by a displaced apex beat and a soft blowing murmur at the mitral area. The first and second sounds were approximate in their intensity. The spleen was just palpable/

palpable (therefore slightly enlarged) and the liver could be felt one finger's breadth below the right costal margin. The knee and ankle jerks were fairly brisk but not unduly so. The plantar reflex gave a flexor response. There was pain in the calf muscles which was exaggerated by squeezing them, but there was no exaggerated tonus. The gait one might describe as rather slow and purposeful - when the patient was not asked to hurry - but was certainly not ataxic.

Examination of the rectum revealed the presence of prolapsed haemorrhoids of moderate degree. There was no free blood in a specimen of stool examined, but the benzdine test gave a triple positive result.

Nothing else was noted at examination and a blood count gave the following result:-

2.11.36.

Haemoglobin.	40.
R.B.C.	2.970.000.
C.I.	0.68.
Halometer reading.	7.2 u.
W.B.C.	6.200.

The stained film showed a moderate degree of anisocytosis but, on the whole, the cells looked larger than the colour index suggested. No primitive or nucleated red cells were seen, but there were quite a few polychromatic cells and a few poikilocytes. The patient was advised about his dietary requirements and given a prescription for ferrous sulphate gr. iii tablets

He/



He was advised to take two tablets thrice daily. He was also advised to take liquid paraffin for the bowels which had been obstinately constipated.

After four weeks the blood showed a definite increase in the haemoglobin but the percentage of cells had not altered. viz.

8.12.36.

Haemoglobin.	60.
R.B.C.	2.770.000.
C.I.	1.09.
Halometer reading.	7.2 u.

The film showed the same polychromasia. There was no obvious degree of anisocytosis or poikilocytosis. No nucleated cells were seen. Apart from the polychromasia, the film might have been that of a healthy individual. I explained the failure of red cell regeneration by further haemorrhage by the bowel, though the patient said he had not been aware of this. Clinically, he was improved. The numbness and paraesthesia had gone. There was no icterus.

There was no clinical evidence of a pernicious anaemia complex. The high colour index, with low cell count, I put down to haemorrhage, as the blood film did not suggest a megaloblastic anaemia. I advised the patient to continue taking the pills and instructed him once more about the vitamin lack in his diet. He had not taken a single orange despite my advice! I found out six months later that he had only taken 85 of the originally/

originally prescribed pills! I saw him one month later, under the impression that he was still taking Ferrous sulphate gr. xviii, daily. The blood picture was:-

3.2.37.

Haemoglobin.	58.
R.B.C.	3.350.000.
C.I.	0.87.
Halometer reading.	7.3 u.

This time he told me he had had a haemorrhage from the bowel, and examination revealed that the haemorrhoids were severely prolapsed and ulcerated. There was no rectal neoplasm. The general health was improved. There were no petechial haemorrhages and his appetite was much better. One month later the following was noted:-

4.3.37.

Haemoglobin.	64.
R.B.C.	3.610.000.
C.I.	0.89.

It was then decided, rather illogically, and with a certain amount of desperation, to treat him as a case of pernicious anaemia. He was advised to carry on with the pills. At the same time anahaemin was exhibited. I left the practice shortly afterwards for six months, and did not see him until September 1937. His treatment in the interval was.

5.3.37.  
29.3.37.  
9.4.37.  
9.5.37.  
7.6.37.  
4.7.37./

2 c.c. Anahaemin.
do. do.
do. do.
do. do.
do. do.

4.7.37.	2 c.c. Anahaemin.
1.8.37.	do. do.
29.8.37.	do. do.
26.9.37.	do. do.

On seeing him on my return I found he had taken in all, only 85 ferrous sulphate tablets. His general condition was slightly improved from the first consultation. There was a severe degree of prolapsed ulcerated haemorrhoids with a triple positive benzidine. The blood examination showed:-

<u>12.10.37.</u>	
Haemoglobin.	55.
R.B.C.	2.750.000.
C.I.	1.00.

The blood picture was taken to be one of recent haemorrhage. No more liver extract was given. Ferrous sulphate gr. vi, t.i.d. was prescribed and I saw personally that the patient took them! Three weeks later the picture was:-

<u>27.11.37.</u>	
Haemoglobin.	51.
R.B.C.	3.040.000.
C.I.	0.85.
Halometer reading.	6.9 u.
W.B.C.	5.200.

The patient refused a surgical opinion concerning the prospects of radical removal on the pile bearing area. He took his pills now, however, with a most punctilious regularity. A further blood count showed:-

<u>15.1.38.</u>	
Haemoglobin.	58.
R.B.C.	3.900.000.
C.I.	0.74.
W.B.C.	7.200.

and/

and later,

13.2.38.

Haemoglobin.	65.
R.B.C.	3.760.000.
C.I.	0.87.
W.B.C.	5.800.

This was approximately the same as he was in February one year previously. Curiously, he was looking and feeling much better and carried out his daily duties very well.

I can make nothing more of him, and it has always been to me a source of wonderment that he allowed me to do the blood counts that he did, considering how resentful and suspicious he was about any innovation, even a simple recommendation about his diet.

Case 75. Mrs O. aet 58.

This was a pale faced woman who was considerably overweight. The menopause had occurred ten years previously. She had had one child, and lived in a very comfortable house with her husband, who owns an interior decorator's business. She was inclined to be self-centred and to magnify any trivial symptom she could produce. She said she felt listless, and easily tired. She thought she required a tonic. On examination it was noted that she had a markedly pale complexion. The conjunctivae were moderately pale.

Nothing else was noted on physical examination, and/



and a blood count revealed the following:-

14.11.36.

Haemoglobin.	95.
R.B.C.	5.130.000.
C.I.	0.93.
Halometer reading.	7.4 u.
W.B.C.	7.300.

I prescribed Ferrous sulphate gr. iiii, t.i.d. and examined her blood from a point of interest four weeks later. The following was noted:-

9.12.36.

Haemoglobin.	93.
R.B.C.	5.110.000.
C.I.	0.91.
Halometer reading.	7.3 u.

I saw her two months later and as she wanted to know "what condition her blood was in" I again examined her and found the following:-

18.2.37.

Haemoglobin.	108.
R.B.C.	5.350.000.
C.I.	1.01.

She was one of the few patients who seemed to enjoy having a blood count performed.

Case 76. William G. aet 58.

This patient was a manual labourer. I had occasion to examine him to rule out the possibility of a specific urethritis. In the course of routine examination I detected the presence of a very slightly enlarged spleen. (One finger's breadth below the left costal margin). He had no complaints other than dysuria/

dysuria, and a burning sensation at the urinary meatus for some time after each act of micturition. There was no clinical evidence of anaemia and an examination of the blood revealed the following:-

14.11.37.

Haemoglobin.	103.
R.B.C.	5.330.000.
C.I.	0.97.
Halometer reading.	7.3 u.
W.B.C.	10.400.

The differential count showed the following:-

Polymorphs.	67	%.
Lymphocytes.	22	".
Mononuclears.	7	".
Eosinophils.	3	".
Basophils.	1	".

Case 77. Thomas H. aet 59.

This patient had been the subject of repeated sore throats in early adult life. He said he had had "kidney disease" when a child, but could not remember whether it had been preceded by any of the exanthemata. I saw him during an attack of subacute nephritis. The urine was scanty and contained albumin and occult blood. The blood pressure was raised 160/104. He responded well with rest in bed and a suitable low protein diet. Two months later when the urinary findings were negative and the blood pressure was still raised - 148/90 - I was struck by his pallor. He said he had always been 'a large pale man,' and he presumably possessed the classical/

classical 'large pale kidneys.' A blood examination was done and the following noted:-

14.2.37.

Haemoglobin.	79.
R.B.C.	4.870.000.
C.I.	0.81.

He said he had always been in the habit of taking "tincture of steel" drops when he was feeling 'blood-less.' He would take 15 - 20 drops, three times a day, and he found that this acted as a tonic. I prescribed Ferrous sulphate gr. iii, t.i.d. but I have not seen him again.

Case 78. Mrs M. aet 60.

This patient was a frail little lady, the mother of four children. I met her fortuitously at the confinement of her daughter, and noticing her marked pallor, arranged for her to have a blood test.

The menopause had occurred four years ago. She said that she felt vaguely 'out of sorts' but was not "bad enough to have the doctor." When pressed to detail her symptoms she said that she had been very easily tired, and had lacked energy for the past two years. She had been breathless on exertion for a similar period. She had noticed lately that she frequently had an attack of palpitations if she went up a hill too quickly. She had a very poor appetite and when/

when directly questioned, she said she had experienced some difficulty in swallowing. She had no sore tongue. She complained vaguely of indigestion, and said her food "lay like a lump of lead in her stomach, for hours after she had eaten it." She suffered from constipation and had to take aperients regularly.

On examination it was noted that she was a pale-faced, thin woman. The mucous membranes of the lips, gums and palate were pale. The tongue was furred, but was neither painful nor atrophic. The finger nails were cracked and brittle although not definitely spoon-shaped. The pulse rate was 88 per minute and the rhythm was regular. The blood pressure was 128/70. Nothing else was made out at physical examination. The blood figures were:-

1.2.37.

Haemoglobin.	63.
R.B.C.	4.690.000.
C.I.	0.68.
Halometer reading.	6.9 u.

I prescribed Ferrous sulphate gr. iii, t.i.d. and after three weeks she was looking and feeling much better. The dysphagia had gone and the appetite was improved. She was less dyspnoeic. The blood picture was:-

27.2.37.

Haemoglobin.	86.
R.B.C.	4.870.000.
C.I.	0.89.
Halometer reading.	7.0 u.

She/



She failed to report again.

Case 79. Daniel B. aet 62.

This patient has for many years suffered from hyperchlorhydria. He had a gastric ulcer, which had been treated by radical operation ten years ago. I gathered that a gastrectomy had been performed. This had probably been done in view of the possibility, at his age, of malignant degeneration. He still had symptoms of hyperchlorhydria when I saw him. He had not called me in but I was, at the time, attending his wife. He kept himself reasonably symptom free by taking regularly an alkali powder after meals, and adhering to a modified Sippy diet.

On examination it was observed that he was very pale-faced. The mucous membranes were moderately pale. There was nothing more of a positive nature to be made out on physical examination. (I looked particularly for nail changes but saw none).

The blood picture was:-

22.11.36.

Haemoglobin.	72.
R.B.C.	4.750.000.
C.I.	0.78.
Halometer reading.	6.9 u.

Ferrous sulphate, iron and ammon. citrate, and saccharated ferrous carbonate were all exhibited but had to be discontinued on account of gastro intestinal upset.

Case 80. Miss Jessie B. aet 62.

This patient is the sister of Isobel B. aet 85. The Misses B. are spinsters and live alone in a little room behind the drapery store which they manage.

Miss B. looked very pale but had no urgent symptoms. I had simply noticed her pallor when I called on her sister, and arranged to examine her blood.

The patient said she had always been pale but felt 'none the worse of it.' She thought she was quite well for her years and apart from a slight impairment in her appetite, an occasional attack of indigestion, and a shortness of breath on exertion, she felt quite fit. She said she had been treated by her doctor for chlorosis when she was a young woman.

The blood picture was as follows:-

6.2.38.

Haemoglobin.	75.
R.B.C.	5.240.000.
C.I.	0.72.
Haemometer reading.	7.0 u.

I prescribed Ferrous sulphate gr. iiii, t.i.d. and she was amazed at the improvement in her general fitness, which became apparent after she had taken the pills for two weeks.

I have no further blood figures.

Case 81. Mrs H. aet 68.

This patient was a nulliparous widow. Her chief and/

and most urgent symptom was soreness of the tongue.

She had for several years suffered from a chronic progressive form of rheumatoid arthritis, which involved the wrists, knees, and ankle joints. The small joints of the fingers and wrists were chiefly affected and grossly deformed. She was also the subject of an intractably recurring chronic bronchitis, which was usually most troublesome during the winter months. She very rarely went out on account of the crippling nature of her arthritis.

When I saw her in November 1936 her chief complaint was soreness of the tongue. She complained also of marked loss of appetite and a feeling of "something sticking in her throat." She had dyspnoea on exertion but as she rarely went about this was not urgent. On examination it was observed that she was a frail little lady who looked considerably older than her years. She had a clear transparent complexion, which was not unduly pale. The mucous membranes of the mouth were moderately pale and the tongue, in contrast, was red and angry-looking. It was not atrophic but looked rather hypertrophied. There was a thick, yellowish fur towards the posterior part of the dorsum but the tip and edges were clean. The finger nails were cracked and had many longitudinal ridges, but were not concave. Nothing else of note was/

was made out at physical examination. The blood count revealed the following:-

16.11.36.

Haemoglobin.	70.
R.B.C.	4.110.000.
C.I.	0.85.
Halometer reading.	7.1 u.
W.B.C.	9.200.

The blood film showed no gross departure from normal. The cells were approximately of equal size; no primitive forms were seen; and they appeared to be fairly well-filled with haemoglobin. A differential white count was not done. I prescribed Ferrous sulphate gr. iii, t.i.d. and after one month the following was noted:-

4.12.36.

Haemoglobin.	84.
R.B.C.	4.520.000.
C.I.	0.93.
Halometer reading.	7.3 u.

She said the tongue condition was less urgent, but though she was feeling improved in her general health she still occasionally had a mild degree of glossodynia, especially when she took any hot liquids.

I did not see her again for a period of two months during which time she had no therapy. I examined the blood and the following was noted:-

3.2.37.

Haemoglobin.	77.
R.B.C.	4.250.000.
C.I.	0.91.
Halometer reading.	7.2 u.

The/



The tongue condition had again become troublesome. I had been giving Anahaemin empirically for a variety of conditions (especially disseminated sclerosis and X-ray sickness) not obviously related to pernicious anaemia, and decided to try it in this case. She received 1 c.c. the following day. Curiously enough she ceased to complain of a sore tongue about one week later. She has had 1cc. of anahaemin at monthly intervals since. The iron therapy was discontinued. Apart from the absence of glossitis, her general condition, when I examined her one year later was unaltered. The blood findings in the interval were:-

17.2.37.

Haemoglobin.	83.
R.B.C.	4.120.000.
C.I.	1.01.
Halometer reading.	7.3 u.

7.3.37.

Haemoglobin.	80.
R.B.C.	4.510.000.
C.I.	0.89.

10.10.37.

Haemoglobin.	80.
R.B.C.	4.230.000.
C.I.	0.95.

Case 82. Mrs O. aet 82.

This patient is a frail old lady, the mother of seven children. She is bed ridden on account of chronic arthritis. She has a pale sallow, weather-beaten complexion. The skin is tough and grossly wrinkled./

wrinkled. She is troubled with obstinate constipation, which yields only to large daily doses of 'Agarol.' She frequently suffers from severe haemorrhage from haemorrhoids. I saw her after such an attack had subsided.

On examination it was observed that the mucous membranes were very pale. The tongue looked atrophic as was natural in view of her age. The nails were not cracked. No other findings apart from the above were noted. The remainder of the patient's complaints could legitimately be dismissed on the grounds of senility. The blood showed the following:-

22.10.37.

Haemoglobin.	38.
R.B.C.	3.490.000.
C.I.	0.48.
Halometer reading.	6.6 u.
W.B.C.	5.200.

I prescribed Ferrous sulphate gr. iii, t.i.d. This caused so much abdominal discomfort and general upset that I stopped them after three weeks, although the blood showed a definite improvement. viz.

14.11.37.

Haemoglobin.	62.
--------------	-----

In view of her age it was decided unwise to make her life any more uncomfortable, than was strictly and urgently necessary. All active therapy was consequently stopped.

The remaining seven cases are subjects of pernicious/

pernicious anaemia. Four of them occurred in the practice where the above work was done. The remaining three came under my notice while a house physician during April - September, 1936. Only the blood counts in this period, therefore, are my own personal observations. The proper authorities have been approached and permission has been granted to use these figures. I desire to use them, in conjunction with my own cases in private practice, to illustrate some points in the follow up treatment of pernicious anaemia.

Case 83. William D. aet 69.

This patient is a retired stone mason. He is a widower, and lives with his dog alone in a small cottage. He does his own cooking, washing and house-work, reads a great deal, and takes a great interest in the local municipal government. He was first seen by Professor G. L. Gulland in March, 1928, and a diagnosis of pernicious anaemia was established. He was fed on raw liver, and sent home from hospital, on a maintenance dose, when his blood count had reached a satisfactory level. He failed to maintain the improvement, however, and his count on April 1930 was:-

Haemoglobin.	60.
R.B.C.	2.680.000.

(Royal Infirmary of Edinburgh).

He was seen again on 30th June, 1931 when his figures were:-

30.6.31.

Haemoglobin.	90.
R.B.C.	4.600.000.

He had a bilateral inguinal hernia and had one side/

side operated on in the local hospital in 1931.

He has since been taking  $\frac{1}{2}$  lb. raw liver three times a week, but failed to report and has not had a blood count done since 1931.

I first saw him in October, 1937. He said he was feeling quite well but was not quite so "strong in the legs" as he used to be. He was not working now, and was never unduly breathless. He was leading a quiet retired life. Both knee jerks were rather brisk but apart from that the entire examination of the central nervous system yielded negative results. There was no splenic or hepatic enlargement. There was no icterus. The face and mucous membranes of the eyes and mouth were pale. On 23rd October, 1937, the blood picture was as follows:-

23.10.37.

Haemoglobin.	68.
R.B.C.	3.270.000.
C.I.	1.05.
Halometer reading.	7.8 u.

Anisocytosis was marked. Poikilocytosis and macrocytosis were noted. Polychromasia and punctate basophilia were slight. Three nucleated red cells and one cell of the erythroblast type were seen.

I suggested that he take more liver, and he promised to take  $\frac{1}{2}$  lb. of ox liver daily. As he is well known to the local butcher he usually gets fair measure for his money, so he is now taking about 4 lbs. of liver per week. He broils this, after mincing it, and/



and can take it practically raw. He appears to have acquired a liking for this rather unpalatable dish.

He was feeling much better when seen about one month later and his blood count showed the following on 6th December, 1937.

6.12.37.

Haemoglobin.	86.
R.B.C.	4.210.000.
C.I.	1.02.

There was quite a marked polychromasia in the stained film. I encouraged him to continue on the increased amount of liver, and after six weeks I examined his blood once more:-

27.1.38.

Haemoglobin.	90.
R.B.C.	5.450.000.
C.I.	0.83.

He was feeling greatly improved and apparently in excellent health. I noted his blood pressure, which was 130/82.

Case 84. Mrs J. B. aet 71.

This patient gave a history of headaches, irritability, sleeplessness, and dyspnoea on exertion, for a period of ten years. She said she had suffered from "blood pressure" but had lately felt much worse.

She had four children, who are all grown up and alive and well. She lives alone with her husband in a three-roomed house, and between them they do all the house-work/

housework. This is truly amazing in view of the data quoted below, and in view also of the fact that the husband has an impossibly irregular pulse as the result of a coronary thrombosis two years previously.

When I first saw her the patient was in a dazed condition and aphasic. Her blood pressure was at that time 220/134. She had however, no evidence of paresis, and the reflexes motor and sensory were normal in both upper and lower limbs. She presented nothing else of note except an oligouria, a slow bounding pulse, and a heaving, displaced, apex beat, with a markedly accentuated aortic second sound. In the course of three or four days she was back to what she called "her usual" and the attack of what the Americans describe as "hypertensive encephalopathy" had subsided. Her "usual" however, I soon discovered, was a very indifferent standard of health. She was very pale; her mucous membranes were blanched. She had a definite icteric tinge, which she put down to her usual sallow complexion. She was troubled with indigestion, flatulence, and anorexia. The least exertion caused her to be extremely dyspnoeic, and evidenced the onset of "palpitations." She was very irritable and suffered from insomnia and frequent headaches. She also said that she had ulcers in her mouth. She admitted that all these complaints had become/

become worse in the last two years, but put them down to "old age!"

Examination revealed the fact that she had no ulcers in the mouth but had a "raw beef" tongue with smooth atrophic tip and edges. There was no evidence of any permanent involvement of the central nervous system, apart from that mention above. The examination of her blood revealed the following:-

26.10.36.

Haemoglobin.	52.
R.B.C.	1.990.000.
C.I.	1.33.
Halometer reading.	7.8 u.
W.B.C.	5.100.

The stained film showed marked anisocytosis and poikilocytosis. Polychromasia was in evidence in a few cells and there was obvious macrocytosis in some parts of this slide. No nucleated red cells were seen, but three cells of the "erythroblast" class were seen. The diagnosis of pernicious anaemia was made.

The progress of the case was as follows:-

26.10.36.

Haemoglobin.	52.
R.B.C.	1.990.000.
C.I.	1.33.
Halometer reading.	7.8 u.
W.B.C.	5.100.

5.11.36.

2 c.c. Anahaemin.

19.11.36.

do. do.

3.12.36.

Haemoglobin.	82.
R.B.C.	5.120.000.
C.I.	0.82.

She/

She was feeling and looking much better. The appetite was greatly improved. She was sleeping much better, had less dyspnoea, and really felt fit for her household duties. She still has headaches if she stoops. The blood pressure was 220/130. She thought she should have no more treatment as she was "just in perfect health." Further notes were as follows:-

4.12.36.	2 c. c. Anahaemin.
18.12.36.	2 do. do.
16.1.37.	1 do. do.

23.2.37.

Haemoglobin.	96.
R.B.C.	5.200.000.
C.I.	0.92.

I prescribed Ferrous sulphate gr. iii, t.i.d. as well as the liver extract, in view of the low colour index.

27.2.37.	1 c. c. Anahaemin.
12.3.37.	1 do. do.
2.4.37.	1 do. do.
29.4.37.	1 do. do.
27.5.37.	1 do. do.
24.6.37.	1 do. Stop Fe. sulph
22.7.37.	1 do. Anahaemin.
19.8.37.	1 do. do.
16.9.37.	1 do. do.
21.10.37.	1 do. do.

21.10.37.

Haemoglobin.	94.
R.B.C.	4.490.000.
W.B.C.	7.200.
C.I.	1.06.

She was feeling full of "joie de vivre" and, apart from occasional headache, had no complaints.  
She/



She lives very quietly with her husband, and does not go out much. She is visited frequently, however, by her family who live in the neighbourhood. She takes a great interest and pride in her grandchildren. Her daughter comes once a week to do the laundry, and the patient and her husband usually do the ironing in the evening. Truly a remarkable couple!

10.11.37.	1 c. c. Anahaemin.
8.12.37.	1 do. do.
4.1.38.	1 do. do.

4.1.38. The blood pressure is 260/148. She is bordering once more on a cerebro vascular catastrophe. She refuses point blank to lead a more restricted life. I prescribed bromides and chloral three times a day, with luminal gr. i, at bed-time.

24.1.38. She is now more amenable to suggestion and under the influence of sedatives, is leading a more restricted existence. The blood pressure is 210/130.

Case 85. James N. aet 76.

This patient is an old retired labourer and is the local kirk-beadle. He says that he took ill in February, 1932, when he had vomiting and diarrhoea. He says that for a long time he had had "pins and needles" in his arms and legs and had always been very pale. He was very short of breath. This gradually became worse, until he had to give up work. He finally/

finally collapsed and thought he was going to die. His wife says he was very "jaundiced" at this time.

He was seen by a specialist and pernicious anaemia was diagnosed. He was given Hepatex orally. A blood examination was made at this time, but there is no record of it. No further blood examination was made. He took the Hepatex, under his doctor's guidance, as he required it, taking the minimum necessary as it was so expensive. When he got "short of breath or rundown" he took more. He averaged about 4 ounces per week, but had lately been taking less as the price was so prohibitive.

I first saw him in November 1936. He was pale but not unduly so for an old man. He was rather deaf and his mental processes seemed to be very slow. His eyesight was poor, due to senile cataract. His movements were slow and his gait had a curious "shuffling" character. His mucous membranes were very pale but the conjunctivae themselves were thick, oedematous, and injected from a chronic conjunctivitis.

He said he was not unduly breathless, and, like all people of his age, strongly resented any suggestion which might be interpreted by him as a sign of approaching senility. He still carried on his duties as kirk-beadle although he admitted that they were at times "a bit of a bother" to him.

His/

His blood pressure was 156/92. His heart and lungs appeared to be healthy. There was no hepatic or splenic enlargement. His knee and ankle jerks were very brisk. The plantar reflex response was indefinite. He had numbness and paraesthesia of the lower limbs, which, however, was only slight in extent. I failed to satisfy myself that there was any sensory loss. He had a very atrophic tongue and there was a slight icteric tinge.

The blood picture was as follows:-

3.11.36.

Haemoglobin.	70.
R.B.C.	2.400.000.
C.I.	1.46.
Halometer reading.	8.1 u.
W.B.C.	4.600.

Macrocytosis was marked. Anisocytosis and poikilocytosis were also seen. The progress notes are as follows:-

10.11.36.	1 c.c. Anahaemin.
1.12.36.	do. do.

10.12.36.

Haemoglobin.	85.
R.B.C.	4.100.000.
C.I.	1.04.
Halometer reading.	7.7 u.

21.12.36.	1 c.c. Anahaemin.
12. 1.37.	do. do.
2. 2.37.	do. do.
23. 2.37.	do. do.
16. 3.37.	do. do.
13. 4.37.	do. do.
1. 5.37.	do. do.
27. 5.37.	do. do.
19. 6.37.	do. do.
6. 7.37.	do. do.
3. 8.37.	do. do.
28. 9.37./	

28.9.37.

1 c.c. Anahaemin.

26.10.37.

do. do.

When I returned to the practice in October,  
I examined his blood and found the following:-

27.10.37.

Haemoglobin.	78.
R.B.C.	3.730.000.
W.B.C.	5.000.
C.I.	1.05.
Halometer reading.	7.4 u.

Anisocytosis was very marked. Polychromasia and  
punctate basophilia were slight in extent. Poikylo-  
cytosis was marked. I decided to change the liver  
extract and gave him Neo Hapatex.

16.11.37.

4 c.c. Neo Hapatex.

After three weeks the following was noted:-

7.12.37.

Haemoglobin.	84.
R.B.C.	4.770.000.
Halometer reading.	7.4 u.
C.I.	0.88.

He was feeling very much better than he did one  
month ago.

7.12.37.

4 c.c. Neo Hapatex.

28.12.37.

Haemoglobin.	83.
R.B.C.	4.430.000.
C.I.	0.94.

28. 12.37.

4 c.c. Neo Hapatex.

18.1.38.

Haemoglobin.	89.
R.B.C.	4.410.000.
C.I.	1.01.

18.1.38.

4 c.c. Neo Hapatex.

I gave/



I gave him Ferrous sulphate gr. iii, t.i.d. but he could not tolerate these and had to give up after two weeks. The blood picture after a further period of four weeks was:-

15.2.38.

Haemoglobin.	99.
R.B.C.	4.420.000.
C.I.	1.13.

There had, therefore, been a certain degree of iron deficiency as well.

The patient is now receiving 4 c.c. Neo Hepatex at four-weekly intervals, and is surprisingly fit for his years.

Case 86. Isobel B. aet 67.

This patient is the sister of Jessie B. case 80. She was diagnosed as a case of pernicious anaemia in 1932. I wrote to the hospital to which she was sent but could obtain no details about treatment. No notes were available, but in her case sheet I came across a letter from the specialist in charge of her, to my principal, stating the blood picture as follows:-

26.10.32.

Haemoglobin.	84.
R.B.C.	3.390.000.
C.I.	1.25.
W.B.C.	7.400.

A note on the film was to the effect that there was slight anisocytosis and poikilocytosis. With the help of the local chemist I traced her treatment back to/

to April 1935. From April 1935 until December 1936, she received Pernaemon forte 2 c.c. every four weeks. From December 1936 until I examined her in November 1937 she had been receiving Anahaemin 1 c.c. every four weeks. No blood examination had been done in the interval.

At the time of examination she appeared to be in fairly good health. She had no complaints. All that could be made out by physical examination was the obvious pallor of the face; a fair degree of pallor of the mucous membranes; the smooth atrophic tongue; and the fine soft silvery white hair.

(James N., the preceding case, had especially fine silvery white hair, which gave him quite a striking appearance.)

The blood picture was as follows:-

11.11.37.

Haemoglobin.	94.
R.B.C.	4.330.000.
C.I.	1.09.
Halometer reading.	7.6 u.
W.B.C.	6.000.

The film showed macrocytosis and a slight degree of poikilocytosis and anisocytosis.

The patient was advised to keep on this same dose of liver extract, i.e. Anahaemin, 1 c.c. four-weekly, by intra muscular injection. I made one other blood examination which showed the following:-

6.2.38. /

6.2.38.

Haemoglobin.	90.
R.B.C.	4.710.000.
C.I.	0.96.

Although she had no central nervous system involvement, in view of the fact that she was still a comparatively young woman at 67, I decided to attempt to raise the red cell to the 5.000.000 mark. I decreased the interval between each injection to two weeks, but I have not yet any further figures on the case.

The following three cases were observed in hospital. Only their ages, treatment, and progress notes are given, as I wish to make a point about the depot treatment only.

Case 1. Margaret B. aet 65.

Date.	R.B.C. (millions)	Haemo- globin.	Treatment.
23. 7.36.	3.01.	70.	
25.7.36.	4.06.	70.	
21. 8.36.	3.84.	60.	Campolon 2 c.c.
8. 9.36.	2.88.	60.	approximately
13. 9.36.			every 3rd day.
21. 9.36.	3.52.	53.	
29. 9.36.	3.49.	62.	
20.10.36.	3.32.	60.	Campolon. 2 c.c.
22.10.36.			do. do.
26.10.36.			do. do.
29.10.36.			do. dd.
2.11.36.			do. do.
5.11.36.			do. do.
19.11.36.			do. do.
26.11.36.			do. do.
30.11.36.			do. do.
3.12.36./			

Date.	R.B.C. (millions)	Haemo- globin.	Treatment.	
3.12.36.			Campolon.	2 c.c.
8.12.36.	3.62.	78 .	do.	do.
10.12.36.			do.	do.
14.12.36.			do.	do.
17.12.36.			do.	do.
21.12.36.			do.	do.
24.12.36.			do.	do.
28.12.36.			do.	do.
4. 1.37.			do.	do.
7. 1.37.			do.	do.
13. 1.37.			do.	do.
14. 1.37.			do.	do.
18. 1.37.			do.	do.
21. 1.37.			do.	do.
25. 1.37.			do.	do.
1. 2.37.			do.	do.
5. 2.37.			do.	do.
8. 2.37.			do.	do.
15. 2.37.			do.	do.
18. 2.37.			do.	do.
22. 2.37.			do.	do.
25. 2.37.			do.	do.
1. 3.37.			do.	do.
4. 3.37.			do.	do.
8. 3.37.			do.	do.
15. 3.37.			do.	do.
18. 3.37.			do.	do.
22. 3.37.			do.	do.
26. 3.37.			do.	do.
29. 3.37.			do.	do.
8. 4.37.	1.98.	58.	do.	do.
12. 4.37.			Anahaemin	1 c.c.
14. 4.37.			do.	do.
19. 4.37.			do.	do.
20. 4.37.	2.37.	65.		
23. 4.37.			do.	do.
26. 4.37.			do.	do.
29. 4.37.			do.	do.
3. 5.37.			do.	do.
6. 5.37.			do.	do.
8. 5.37.	4.71.	86.		
13. 5.37.			do.	2 c.c.
16. 5.37.	4.73.	84.		
16. 6.37.			do.	do.
18. 6.37.	4.10.	84.		
16. 7.37.			do.	do.
20. 7.37.	3.95.	80.		

Thereafter 2 c.c. Anahaemin once a month  
and no other therapy.

8. 1.38. 4.48. 65.

This patient received from 20.10.36. until

8.4.37., 37 x 2 c.c. Campolon, and her R.B.C. level

fell/



fell throughout it from 3.320.000 to 1.980.000.

From 20.4.37. until 8.5.37. she received 8 x 1 c.c. Anahaemin, and the red cells rose from 1980.000 to 4.710.000. Thereafter she received 2 c.c. Anahaemin at monthly intervals, and eight months later the red cell level was still fairly satisfactory -4.480.000 (8.1.38.).

Case 2. Janet S. aet 74.

This patient had Hepatex daily till 10.7.35. and at the same time, 2 c.c. Campolon every second day while in hospital.

<u>Date.</u>	<u>R.B.C.</u> <u>(millions)</u>	<u>Haemo-</u> <u>globin.</u>	<u>Treatment.</u>
17.5.35.	1.13.	28.	
20.5.35.	1.40.	32.	
27.5.35.	1.31.	35.	
9.6.35.	1.49.		
8.7.35.	2.02.	54.	
3.8.35.	3.79.	78.	
19.8.35.	3.72.	72.	
29.8.35.	3.92.	82.	
17.9.35.	3.60.	79.	
Discharged from hospital. Readmitted on 6.7.37.			
8. 7.37.	1.33.	24.	4 c.c. Anahaemin.
31. 7.37.	1.73.	32.	do. do.
17. 8.37.	2.13.	42.	
19. 8.37.			do. do.
28. 8.37.			do. do. Start
			Ferri. et ammon. cit.
			gr. xx, t.i.d.
10. 9.37.			4 c.c. Anahaemin.
25. 9.37.	3.50.	70.	Ferri. et ammon. cit.
			gr. xx, t.i.d.
13.10.37.			Has had no more Anahaemin

From May till September 1935, she had Campolon  
2 c.c./

2 c.c. every second day, i.e. approximately 60 x 2 c.c. Campolon.

From 8.7.37. till 25.9.37. she had 5 x 4 c.c. Anahaemin.

The red cell level rose in the second period from 1.330.000 to 3.500.000, but this was a period of only two months, as opposed to a period of four months in the Campolon series.

Case 3. Thomas H. aet 80.

<u>Date.</u>	<u>R.B.C.</u> <u>(millions).</u>	<u>Haemo-</u> <u>globin.</u>	<u>Treatment.</u>
24.12.36.	1.20.	30.	
27.12.36.			10. c.c. Campolon.
28.12.36.	1.10.	30.	
30.12.36.			10 c.c. do.
31.12.36.			5 c.c. do.
1. 1.37.			5 c.c. do.
4. 1.37. until 24.3.37.			5 c.c. do.daily
21. 1.37.	1.82.	32.	
6. 4.37.	3.34.	70.	
17. 4.37.			2 c.c. Anahaemin
19. 4.37.			2 c.c. do.
20. 4.37.	4.94.		

From 27.12.36. until 24.3.37. he received 86 x 5 c.c. Campolon and the blood failed quite to reach a satisfactory level. From 6.4.37. until 20.4.37. he received 2 x 2 c.c. Anahaemin and the red cells rose from 3.340.000 to 4.940.000. He was discharged from the hospital soon afterwards and he failed to report.

Index showing case number and page on which it is found.

<u>Case Nos.</u>	<u>Page.</u>	<u>Case Nos.</u>	<u>Page.</u>
1.	9.	44.	78.
2.	9.	45.	82.
3.	10.	46.	84.
4.	11.	47.	86.
5.	12.	48.	87.
6.	14.	49.	88.

# SUMMARY OF FINDINGS

## AND CLINICAL CLASSIFICATION OF 68 CASES OF ANAEMIA.

7.	17.	50.	93.
10.	20.	51.	95.
11.	20.	52.	96.
12.	22.	53.	97.
13.	23.	54.	99.
14.	24.	55.	100.
15.	26.	56.	102.
16.	27.	57.	103.
17.	28.	58.	104.
18.	30.	59.	105.
19.	32.	60.	106.
20.	33.	61.	108.
21.	34.	62.	109.
22.	36.	63.	111.
23.	38.	64.	112.
24.	41.	65.	113.
25.	43.	66.	114.
26.	45.	67.	115.
27.	46.	68.	116.
28.	47.	69.	118.
29.	51.	70.	119.
30.	52.	71.	120.
31.	54.	72.	121.
32.	56.	73.	122.
33.	58.	74.	123.
34.	59.	75.	124.
35.	60.	76.	125.
36.	67.	77.	126.
37.	69.	78.	127.
38.	70.	79.	128.
39.	71.	80.	129.
40.	73.	81.	130.
41.	75.	82.	131.
42.	77.	83.	132.
43.	78.	84.	133.
		85.	134.
		86.	135.
		87.	136.
		88.	137.
		89.	138.
		90.	139.
		91.	140.
		92.	141.
		93.	142.
		94.	143.
		95.	144.
		96.	145.
		97.	146.
		98.	147.
		99.	148.
		100.	149.

Index showing case number and page on which it is found.

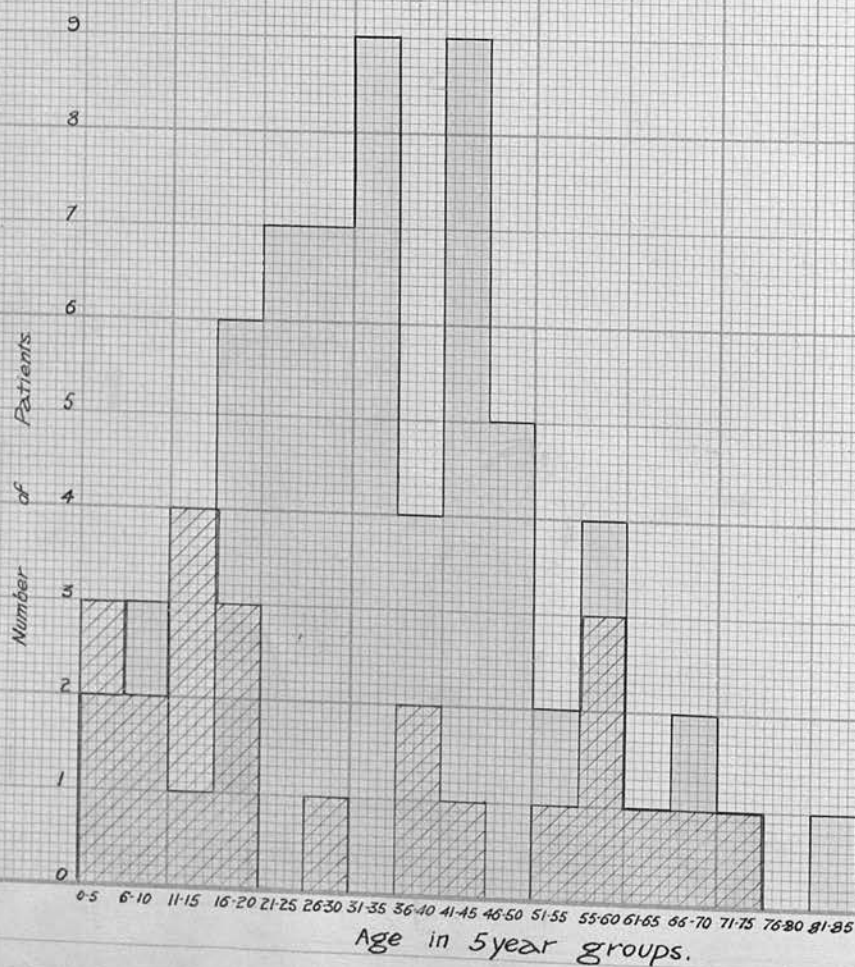
<u>Case Nos:</u>	<u>Page.</u>	<u>Case Nos:</u>	<u>Page.</u>
1.	9.	44.	78.
2.	9.	45.	82.
3.	10.	46.	84.
4.	11.	47.	86.
5.	13.	48.	87.
6.	14.	49.	88.
7.	15.	50.	89.
8.	16.	51.	90.
9.	17.	52.	93.
10.	20.	53.	95.
11.	20.	54.	96.
12.	22.	55.	97.
13.	23.	56.	99.
14.	24.	57.	102.
15.	26.	58.	103.
16.	27.	59.	104.
17.	28.	60.	105.
18.	30.	61.	107.
19.	32.	62.	108.
20.	33.	63.	108.
21.	34.	64.	111.
22.	36.	65.	111.
23.	38.	66.	113.
24.	41.	67.	114.
25.	42.	68.	115.
26.	43.	69.	116.
27.	45.	70.	116.
28.	49.	71.	117.
29.	51.	72.	119.
30.	52.	73.	120.
31.	54.	74.	121.
32.	60.	75.	128.
33.	62.	76.	129.
34.	65.	77.	130.
35.	66.	78.	131.
36.	67.	79.	133.
37.	68.	80.	134.
38.	70.	81.	134.
39.	71.	82.	137.
40.	73.	83.	139.
41.	75.	84.	141.
42.	77.	85.	145.
43.	78.	86.	149.



# Graph shewing number of patients examined arranged in age groups. Total 86.

Fig.1.

MALE IN RED  
FEMALE IN BLUE



Graph shewing the distribution in age groups of 200 patients appearing consecutively at the surgery for any condition.

MALE IN RED  
FEMALE IN BLUE

Fig 2.





(Fig 1) The blood of 86 individuals, varying in age from 3 to 82 years, was examined. These cases fall into three groups.

(1). Those who present a clinical syndrome very suggestive of anaemia -

46 cases - of whom only 2 were not anaemic.

(2). Those presenting vague symptoms or signs which might indicate an anaemia, -

30 cases - of whom 8 were not anaemic.

(3). Cases I did as controls, expecting to find normal values -

10 cases - of whom 2 were anaemic.

Subtracting, therefore, the 18 cases who were not anaemic, we are left with 68 cases of anaemia. These I have arranged in age groups, as follows,

Pre school age.	2 cases.
School children.	12 cases.
Adolescents.	1 case.
Adult males.	7 cases.
Adult females.	46 cases.
Total.	68 cases.

These are arranged graphically. I also prepared a graph showing the age and sex incidence of 200 patients seen consecutively in the surgery. (Fig. 2).

It will be observed from this graph, which can be taken as a rough average of the age incidence of the various patients seen in routine work, that,

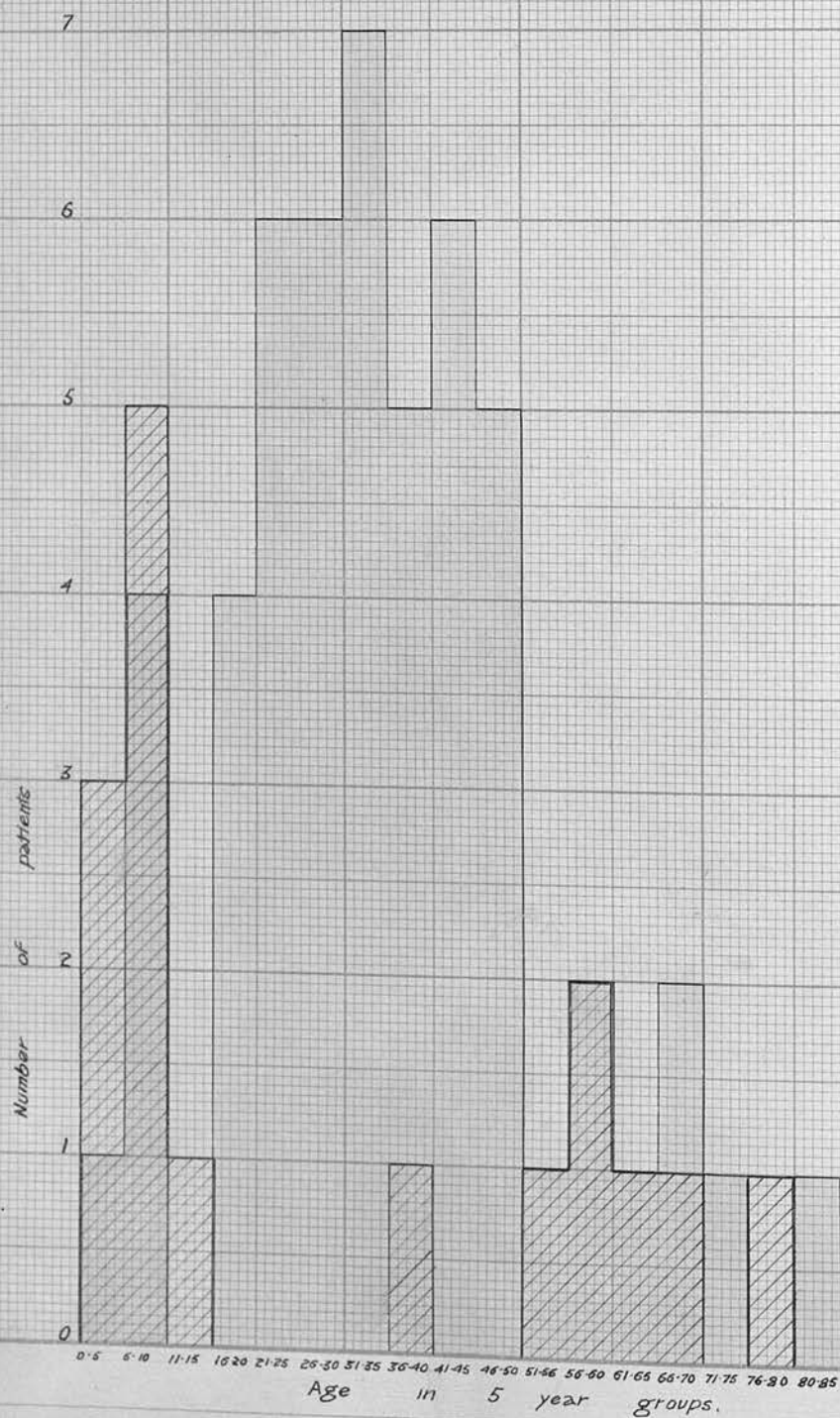
(1). the highest percentage of incidence of both sexes is in the 16 to 20 age group.

(2). the graph for males falls away gradually, and/

Graph shewing distribution in age groups of 68 cases of Anemia.

MALE IN RED  
FEMALE IN BLUE

Fig.3





and after 55 years has dropped to the 4% mark.

- (3). Further, the graph for females is not nearly so sustained, and falls away very abruptly after the age of 30.

The second graph/showing the incidence of anaemia (Fig.3) is a reversal of the first. There is an obvious preponderance of females, and the peak is shifted to the right. The maximum incidence is in the 31 - 35 age group. Although a large number of cases is not available, the evidence is, at least, suggestive that anaemia is common in children and adult women.

The next step is to exclude cases with a definite demonstrable organic basis. I am dealing with the children (14 cases) and adolescents (one case) later. An investigation of the adults (53 cases) reveals the following:-

Males: 7 cases of anaemia.

Pernicious anaemia.	2.
Peptic ulcer.	2.
Haemorrhage.	1.
Nephritis.	1.
Lead poisoning.	1.

Total. 7.

Females: 46 cases of anaemia.

Pernicious anaemia.	2.
Haemorrhage.	2.
Duodenal ulcer.	1.
Associated with pregnancy.	6.
Following deep X-ray therapy.	1.
Focal sepsis.	4.
"Idiopathic."	30.

Total. 46.

Of the 30 cases of idiopathic hypochromic anaemia 4 had been receiving iron therapy before the blood was examined. I propose to study in some detail the findings in the remaining 26 cases.

For purposes of discussion the cases of anaemia are arranged in the following groups.

- (1). Anaemia in children. (14 cases).
- (2). Anaemia in adolescents. (1 case).
- (3). Pernicious anaemia. (7 cases).
- (4). Hypochromic anaemia in adult males. (5 cases).
- (5). Hypochromic anaemia in adult females.  
(44 cases).
- (a). 'Idiopathic' hypochromic anaemias.  
(30 cases).
- (b). Anaemia associated with pregnancy.  
(6 cases).
- (c). Anaemia following haemorrhage. (2 cases).
- (d). Anaemia associated with focal sepsis.  
(4 cases).
- (e). Anaemia associated with duodenal ulcer.  
(1 case).
- (f). Anaemia following deep X- ray therapy.  
(1 case).

Before dealing with the cases in the groups indicated I shall indicate,

1. My technique of doing a blood count.
2. The normal standards for Haemoglobin in health with a note on the various methods of measuring Haemoglobin percentage.
3. The standardisation of my haemoglobinometer.
4. The normal mean diameter for red blood corpuscles and my experience in the use of the Halometer.

The appointments for blood examinations were nearly all made for the early afternoon. The Sahli instrument did not allow of readings being taken in the evening (when I would have had more time to do so) and the forenoon was more suitable for obvious reasons.

MY TECHNIQUE FOR DOING BLOOD COUNTS.

Several of the patients could not, for various reasons, attend, and had to be seen in their own homes. As this entailed the transport of bottles of solutions, pipettes, slides, and a microscope, the patients were instructed to report, wherever possible, at the Surgery. The shipment of apparatus to a patient's home was always rewarded, at least, with a blood count, but many of the cases who were asked to report failed to do so.

One of the earliest observations I made was the increasing depth of colour which occurred with the passage of time, in using the Sahli haemoglobinometer. I decided to adopt an unvarying technique in each case. The red cell diluting fluid was

sodium chloride	1
sodium sulphate	5
corrosive sublimate	0.5
distilled water	200

and the solution for leucocyte was,

glacial acetic acid	1.5
gentian violet 1% sol.	1.0 c.c.
distilled water to	20 c.c.

Two ounce supplies were obtained from the frequent intervals. Jenner's stain was used for examination of red

The appointments for blood examinations were nearly all made for the early afternoons. The Sahli instrument did not allow of readings being taken in the evening (when I would have had more time to do them) and the forenoons were not suitable for obvious reasons. Several of the patients could not, for various reasons, attend, and had to be done in their own homes. As this entailed the transport of bottles of solutions, pipettes, slides, and a microscope, the patients were instructed to report, wherever possible, at the Surgery. The shipment of apparatus to a patient's home was always rewarded, at least, with a blood count, but many of the cases who were asked to report failed to do so.

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sodium chloride	1
sodium sulphate	5
corrosive sublimate	0.5
distilled water	200

and the solution for leucocyte was,

glacial acetic acid	1.5
gentian violet 1% sol.	1.0 c.c.
distilled water to	98 c.c.

Two ounce supplies were obtained fresh at frequent intervals.

Jenner's stain was used for examination of red cell/



cell morphology, and Leishman's for the differential leucocyte count. These were prepared by dissolving Burroughs-Wellcome tabloids in pure methyl alcohol.

All instruments were laid out in readiness. The haemoglobinometer was filled to the mark 10 with N/10 hydrochloric acid. The ball of the thumb of the patient's left hand (in very young children, the heel) was cleansed with rectified spirit and dried with cotton wool. A sharp sudden stab was made with the point of a very fine, sharp cataract knife. The first drop of blood was discarded. The blood was allowed to flow of its own accord without pressure. The haemoglobinometer pipette was filled to the mark, wiped, and the blood blown into the acid and mixed by blowing carefully down the pipette. The thumb was then wiped free of blood and when a fresh drop had collected the red cell pipette was filled to the mark 0.5, the tip wiped dry, and diluting fluid sucked up to the 101 mark. The instrument was rotated to ensure thorough mixing. In most cases a leucocyte pipette was similarly charged.

A blood film was made by the two slides method. The coverslip was placed on the haematocytometer slide and, discarding the first six drops, a drop of diluted blood from the red cell pipette was run under the coverslip. If the diluted solution failed to reach the distal end of the coverslip, or overflowed the platform/

platform, the slip was taken off, the slide cleansed and dried, and another attempt was made.

The acid blood mixture was now diluted drop by drop with distilled water from a pipette until the colours were practically matched. Attention was then directed to the microscope. The haematocytometer slide had, meanwhile, been lying on the stage, giving the erythrocytes time to settle down in their capillary meanderings. The contents of 5 triple ruled squares were counted separately. I always took each corner square and one in the centre to secure a fair average. The cells touching the upper or right hand boundary were always ignored, and those touching the other two sides included in the count. A leucocyte count was prepared and the average of four freshly prepared slides taken.

In the interval of waiting for the leucocyte to settle a Halometer reading was taken and, finally, the data noted in a card index system with the date, the patient's age, sex, and other relevant data.

Finally, the haemoglobinometer reading was taken, only after 30 minutes from the time that the blood was discharged into the acid. The haemoglobinometer reading was noted and the correction made from a graph prepared at a later date. In a doubtful case, or in the case of a leucocytosis, the films used in the Halometer were stained and examined later when circumstances permitted.

The/

The pipettes were then rinsed in succession (by means of a rubber bulb attached to the end) with liquor potassii, distilled water, alcohol and ether, and put away. Slides were cleansed by a similar process and kept in rectified spirit.

THE PRINCIPLES OF HAEMOGLOBINOMETRY.

Normal Standards of Haemoglobin  
in Health.

## Normal Haemoglobin Percentage In Health.

A brief perusal of medical literature reveals a bewildering range of so-called 'normal' standards of haemoglobin level in the blood of healthy human subjects.

## THE PRINCIPLE OF HAEMOGLOBINOMETRY.

Figures published, in regard to the number of haemoglobinometers

### Normal Standards of Haemoglobin.. in Health.

For clinical purposes, haemoglobinometers are all colorimeters in their mode of action. These instruments are standardized with standard solutions of known haemoglobin content, and are calibrated against the more elaborate colorimeters of the laboratory, which are themselves calibrated by spectrophotometric methods.

Although such instruments have reached a pitch of perfection such that their percentage of error is negligible, we are, nevertheless, still 'as one.' They are merely, at their best, comparative solutions of blood, and state, very accurately, the percentage of haemoglobin present in terms of a known standard. This standard may either be:

(1). a series of 'healthy normal' subjects,

whose blood is alleged to contain the normal percentage of haemoglobin, or

(2). a series of cases in which the haemoglobin

percentage/



### Normal Haemoglobin Percentage In Health.

A brief perusal of medical literature reveals a bewildering range of so called 'normal' standards of haemoglobin level in the blood of healthy human subjects.

Almost proportionate to the number of normal figures published, is the number of haemoglobinometers in use.

For clinical purposes, haemoglobinometers are all colorimetric in their mode of action. These practicable bed side instruments are standardised against the more elaborate colorimeters of the laboratory, which are themselves standardised by tedious and complicated physico-chemical methods.

Although modern instruments have reached a pitch of perfection such that their percentage of error is negligible, we are, nevertheless, still 'at sea.' They can merely, at their best, compare solutions of blood, and state, very accurately, the percentage of haemoglobin present in terms of a known standard. This standard may either be,

- (a). a series of 'healthy normal' subjects, whose blood is alleged to contain the normal percentage of haemoglobin, or
- (b). a series of cases in which the haemoglobin percentage/

percentage is known, and has been calculated from first principles.

In theory, there is only one known method of obtaining a series of solution of haemoglobin, the percentage of which is known. That is, by the preparation of a solution of pure haematin crystals.

On the other hand, if we know the iron content or the oxygen combining power of the haemoglobin molecule, two more methods are at our disposal:-

- (a). By estimating the iron content of a sample of blood, or
- (b). by estimating the oxygen combining power of a sample of blood.

Unfortunately as Van Slyke states ( 4 ) in his monumental work,

"The precise ascertainment of the Fe. content of purified human haemoglobin is a problem that awaits solution."

With regard to the first method, namely the preparation of a solution of pure haematin crystals, he quotes the results of Williamson ( 5 ) who used this method of standardising his instrument. He used photometric methods and studied the blood of 919 individuals. Van Slyke, however, doubts the purity of his solution of haematin crystals and states that the results were probably too high.

There remains, therefore, but one final choice, and that is to estimate the oxygen combining power of the blood.

It/

It is very interesting to note that all modern methods revert finally to this estimation.

The classical experiments of Haldane at the end of last century, attain therefore, a particularly significant importance. Haldane's work has coloured - the pun is unintentional - the whole of the work of English investigators in haemoglobinometry. More refined and elaborate techniques now used do not materially differ from his method, as originally published in 1897, of estimating the oxygen combining power of blood. We can be excused from displaying a little pride in our University, when we find that the experiments of Haldane and Lorrain Smith are quoted by all serious investigators of the subject.

So important are these experiments that it is proposed to quote in some detail the original publications.

The underlying principles of Haldane's method of arriving at the oxygen tension in the blood, were described by himself in 1895. ( 6 ). In 1896 Haldane and Lorrain Smith ( 7 ) described a series of experiments whereby they arrived at a figure of 26.2% of an atmosphere for the oxygen tension of arterial blood. They argued as follows:- (p.501).

"Now the final saturation with carbonic oxide (CO) and oxygen depends on the relative tension of carbonic oxide and oxygen in the liquid, so that/



"that if the tension of carbonic oxide, and the final saturation of the haemoglobin be known, the oxygen tension can be inferred."

They also argued that if the carbon monoxide tension of the blood leaving the lungs is known as well as the final saturation of the blood with carbon monoxide, the oxygen tension of the blood leaving the lungs can be calculated.

Since the carbon monoxide tension of the blood leaving the lungs is equal to the tension of carbon monoxide in the alveoli, if the percentage of carbon monoxide breathed is known and the final saturation of the blood is also known (having been estimated colorimetrically against a known solution of carboxyhaemoglobin) the carbon monoxide or oxygen tension of the arterial blood can be calculated.

A further advance was made when Haldane in 1897 described his method of estimating the oxygen combining power of blood. He sums up ( 8 )

"When ferricyanide is added to a solution of oxyhaemoglobin or carbonic-oxide-haemoglobin the gas combined with the haemoglobin is set free and froths off, while methhaemoglobin is formed.

By taking advantage of this reaction the volume of gas capable of being absorbed by the haemoglobin of blood may be rapidly and accurately determined."

Haldane indicated further improvement in his gas analysis technique in the same year. ( 9 ).

Next year ( 10 ) he refuted the idea that carbon monoxide was oxidised in the body. This removed an obvious fallacy in the deductions from his experimental findings. In the same year he published further improvements/



improvements in his ferri cyanide technique ( 11) and shortly afterwards, in collaboration with Lorrain Smith, published the results of his epoch marking experiments on the oxygen combining power of healthy human blood, and the **total** volume of circulating blood. The steps were as follows:-

- (1). The oxygen capacity of fresh ox blood was determined by the ferri cyanide method.
- (2). The oxygen capacity of the patient's blood was calculated by obtaining a drop from a finger prick and comparing it colorimetrically with the standard ox blood to estimate the  $O_2$  capacity.
- (3). The patient was made to breathe a known volume of CO.
- (4). The percentage of CO saturation of the patient's blood was calculated by comparing with a picrocarmine standard.

Since the volume of CO given was known and the percentage saturation was also known, the total volume of CO (or oxygen) capable of being taken up by the blood could be calculated. But the percentage oxygen capacity of the blood was already known so the volume of blood in circulation could be calculated. Knowing the mean specific gravity of blood the total mass could be calculated.

These workers found the percentage oxygen capacity of/

of the blood to be 18.5%.

It is very interesting to note that Haldane in this publication (12) assumes

"that 1 gram of haemoglobin takes up about 1.25 c.c. of oxygen." This would give his figure of 18.5% a reading of 14.8 grammes of haemoglobin per 100 c.c. of blood. This corresponds to 107% on his own scale and is in keeping with the results of the most modern workers. I have not seen this point referred to in any of the textbooks. It is of no importance, however, since Haldane in a later paper adopts Hufner's formula of 1gram Haemoglobin - 1.34 c.c. oxygen and on this basis his 18.5% oxygen capacity represents 13.8 grammes Haemoglobin per 100 c.c. of blood.

In 1901 Haldane checked his normal figures by the ferri cyanide method. It was in this publication that the definition of the Haldane scale was made. He said (13 ).

"I have definitely adopted a standard solution for the haemoglobinometer, a 1% solution of ox or sheep blood of 18.5% oxygen capacity. The exact percentage of haemoglobin corresponding to 18.5% capacity is still uncertain. According to Hufner's latest results it would be 13.8%."

100% on this haemoglobinometer corresponds to an oxygen capacity of 18.5% or 13.8 grammes of Haemoglobin per 100 c.c. of blood. This is what we mean by the Haldane scale.

It is interesting to note that no serious attempt has been made to verify the figure 1.34 which Hufner obtained in 1894. Van Slyke (14 ) states,

"This value is without justification used at the present day to estimate in grammes the haemoglobin content of human blood from analytically determined O<sub>2</sub> or CO combining capacities."

Price Jones (15 ) using the Haldane scale examined the blood, by oxygen combining power, of 100 healthy males/

males and 96 healthy females. He gave the following average readings,

Male.....	Average haemoglobin.	105.42%
Standard deviation. ....		3.92.
Female.....		98.26.
Standard deviation.....		4.42.

He referred to the higher figures given by various American authorities. With healthy male students at Boston, he himself obtained an average reading of 112%. He also stated that the Van Slyke method of obtaining the oxygen combining power of blood gave results "slightly higher but not certainly different from those given by the Haldane ferri cyanide method." One other factor, he suggested, might also be the cause of the rather higher American figures, and that was a mild chronic degree of carbon monoxide poisoning (leading to a compensatory stimulation of erythropoietic activity) brought about by the CO content in the exhaust fumes from motor cars. In a later paper by Price Jones, Vaughan and Goddard (16) figures from 100 healthy English males, estimated by Haldane's method, are given. The average haemoglobin percentages were,

- (a). for venous blood.....105.467 %
- (b). for capillary blood.....106.68 %

Using a similar method Whitby and Hynes (17) in the same year found the average for 50 cases to be:-

Haemoglobin 112% with variation between 100 and 126%.

In the same year, Holiday, Kerridge, and Smith (18) published/



published an account of an elaborately sensitive photo electric method of comparing the concentration of oxyhaemoglobin in solution. The principle of the method is, that the amount of light from a given source which is absorbed in its passage through a chamber of known dimensions, can be measured. Oxyhaemoglobin in solution is placed in the chamber. The other factors being known the concentration of blood can be calculated, as it is directly proportional to the amount of light absorbed. They studied the blood of 'normal' subjects whose blood had been estimated by the Haldane method. They gave a normal figure of:-

Haemoglobin.....105.5%.

Using Haldane's method, Fullerton, Lyall and Davidson ( 19 ) give the following results in the study of 16 normal individuals:-

Average Haemoglobin.	101%
Minimum reading.	93%
Maximum reading.	110%

The above figures are the results of English workers on standard haemoglobin levels in adults.

The figures for children are more confusing. As the question of anaemia in infancy does not arise in this paper I shall confine my attention to figures for children and adolescents. Three authorities are quoted:-

(a). Hutchison in 1904 ( 20 ) gives the following standards in the Haldane scale. At six months/



months the haemoglobin percentage is 70, at which level it remains till the beginning of the second year. Thereafter it steadily rises and reaches the adult standard at the 6th year or later.

(b). Holt and McIntosh in 1933 (21) give the following levels on the Haldane scale:-

<u>Age.</u>	<u>Haemoglobin.</u>	<u>R.B.C.</u>
2 years.	70-80.	4.5.
5 do.	85.	4.5.
10 do.	90.	5.0.

(c). Davidson et. al. 1935 (22) state:-

"A review of the literature afforded little help in regard to Haemoglobin concentration in children and adolescents. We have decided to adopt the following normal standard:-

over 5 months.	86.
2 - 5 years.	90.
school age.	95.
adolescents.	105. "

They state that the normal range is 10% above or below. Anaemia cannot be said to be present unless the figures fall 10% below the normal limit.

#### Summary.

1. A brief description is given of the principles of haemoglobinometry.
2. The original method of Haldane is described.
3. The definition of Haldane's Standard Scale - which is used throughout this paper - is given.
4. Figures for normal healthy individuals are quoted/

quoted from various authorities.

5. I have decided to use the following percentages as 'borderline' normals.

<u>Age.</u>	<u>Haemoglobin per cent.</u>
2 years.	70.
5 do.	80.
12 do.	83.
Adult females.	85.
" males.	95.

I gradually gathered the impression that my haemoglobinometer readings were too high. Although the instrument was of the all-glass, non-fade variety, I had reason to question the readings obtained. These were chiefly:-

(1). THE STANDARDISATION OF MY HAEMOGLOBINOMETER.

Index frequently presented a clinical syndrome, which was highly suggestive of a so called idiopathic hypochromic anaemia.

(2). Taken in conjunction with the clinical findings, the blood picture - appearance of the film and red cell level - would often fit in with such a diagnosis, were it not for the high haemoglobin figure obtained.

(3). In the cases of pernicious anaemia, a very high spleen index was sometimes obtained. Thus the uncorrected values were:-

In case 34

Haemoglobin.	55.
R.B.C.	1,980,000.
C.I.	1.44.

In case 35

Haemoglobin.	75.
R.B.C.	2,400,000.
C.I.	1.58.

The readings were subsequently corrected to (haemoglobin 52 and 70, respectively, on the Sahli scale.) In working out these colour indices I am taking 100 % as normal. It will be observed that in Sahli's original scale of 100 % 15.3 gms. of Haemoglobin per 100 c.c. of blood, (case/

I gradually gathered the impression that my haemoglobinometer readings were too high. Although the instrument was of the all-glass, non-fade variety, I had reason to question the readings obtained. These were chiefly:-

- (1). Patients with a fairly high or normal colour index frequently presented a clinical syndrome, which was highly suggestive of a so called idiopathic hypochromic anaemia.
- (2). Taken in conjunction with the clinical findings, the blood picture - appearance of the film and red cell level - would often fit in with such a diagnosis, were it not for the high haemoglobin figure obtained.
- (3). In the cases of pernicious anaemia, a very high colour index was sometimes obtained. Thus the uncorrected values were:-

in case 84

Haemoglobin.	56.
R.B.C.	1.990.000.
C.I.	1.44.

in case 85

Haemoglobin.	75.
R.B.C.	2.400.000.
C.I.	1.56.

(the readings were subsequently corrected to - haemoglobin 52 and 70, respectively, on the Haldane scale.) In working out those colour indices I am taking 100 % as normal. It will be observed that in Sahli's original scale of 100 % = 17.3 gms. of Haemoglobin per 100 c.c. of blood, these/



these readings would represent, on Haldane's scale (which is normally used in working out colour indices) 70 and 94 % respectively, which would give the ridiculous colour indices of 2.21 and 1.96. Janet Vaughan (23) states that figures higher than 1.4 are probably due to experimental error. As I had adopted a fairly constant technique in each case, I had no reason to doubt that my values for red blood corpuscles were beyond the normal bounds of experimental error, and so the haemoglobin values were rendered suspect.

- (4). A further reason for doubting the haemoglobin values obtained was furnished in case 51. This lady had her blood examined by a specialist a few days after my observations were made. He used a Sahli instrument, and obtained a reading of 68 as opposed to mine of 76 %. He informed me that he took the reading at three minutes, as his standard for working out a colour index.

I prepared a chart from the readings of 20 patients taken at different time intervals, hoping to discover, if the results were uniform, a fixed time interval when the readings were approximately equal to those on the Haldane scale. The cases on which the results are based are indicated. (The last three were from a control/

Time Interval.	Cases,																	x	x	x	
	1. (23)	2. (85)	3. (23)	4. (self)	5. (31)	6. (45)	7. (30)	8. (74)	9. (22)	10. (53)	11. (4)	12. (56)	13. (74)	14. (86)	15. (80)	16. (27)	17. (17)	6.	2.	4.	
1.	63.	63.	71.	83.	68.	68.	65.	46.	75.	80.	61.	56.	54.	71.	58.	62.	70.	43.	82.	70.	65.49. (Average).
3.	72.	81.	78.	88.	75.	75.	80.	52.	82.	90.	70.	62.	59.	79.	70.	74.	77.	52.	92.	76.	
5.	77.	81.	85.	90.	82.	81.	85.	55.	87.	97.	74.	68.	64.	85.	72.	78.	80.	52.	97.	82.	
7.	82.	85.	92.	93.	85.	87.	87.	57.	92.	99.	76.	70.	66.	94.	76.	80.	86.	54.	102.	86.	
10.	86.	88.	94.	96.	87.	89.	92.	58.	94.	103.	80.	72.	70.	96.	78.	83.	90.	57.	105.	88.	
15.	88.	91.	96.	98.	90.	90.	95.	61.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	108.	90.	
20.	90.	95.	98.	103.	90.	90.	97.	63.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	110.	90.	
25.	90.	95.	98.	108.	90.	90.	97.	63.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	110.	90.	
30.	90.	95.	98.	110.	90.	90.	97.	63.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	110.	90.	
35.	90.	95.	98.	112.	90.	90.	97.	63.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	110.	90.	
40.	90.	95.	98.	112.	90.	90.	97.	63.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	110.	90.	88.9. (Average).
Increase.	27.	32.	27.	29.	22.	22.	32.	17.	26.	28.	23.	16.	16.	25.	22.	21.	22.	14.	28.	20.	23.45. (Average).
Time taken to reach max. reading	20.	20.	20.	35.	15.	15.	20.	20.	15.	15.	15.	10.	10.	10.	15.	10.	15.	10.	20.	15.	16.25. (Average).

x. Cases 6, 2, and 4, respectively, of the series of  
six controls quoted below.

Time Interval.	Cases,																	x	x	x	
	1. (23)	2. (85)	3. (23)	4. (self)	5. (31)	6. (45)	7. (30)	8. (74)	9. (22)	10. (53)	11. (4)	12. (56)	13. (74)	14. (86)	15. (80)	16. (27)	17. (17)	6.	2.	4.	
1.	63.	63.	71.	83.	68.	68.	65.	46.	75.	80.	61.	56.	54.	71.	58.	62.	70.	43.	82.	70.	65.49. (Average).
3.	72.	81.	78.	88.	75.	75.	80.	52.	82.	90.	70.	62.	59.	79.	70.	74.	77.	52.	92.	76.	
5.	77.	81.	85.	90.	82.	81.	85.	55.	87.	97.	74.	68.	64.	85.	72.	78.	80.	52.	97.	82.	
7.	82.	85.	92.	93.	85.	87.	87.	57.	92.	99.	76.	70.	66.	94.	76.	80.	86.	54.	102.	86.	
10.	86.	88.	94.	96.	87.	89.	92.	58.	94.	103.	80.	72.	70.	96.	78.	83.	90.	57.	105.	88.	
15.	88.	91.	96.	98.	90.	90.	95.	61.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	108.	90.	
20.	90.	95.	98.	103.	90.	90.	97.	63.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	110.	90.	
25.	90.	95.	98.	108.	90.	90.	97.	63.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	110.	90.	
30.	90.	95.	98.	110.	90.	90.	97.	63.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	110.	90.	
35.	90.	95.	98.	112.	90.	90.	97.	63.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	110.	90.	
40.	90.	95.	98.	112.	90.	90.	97.	63.	101.	108.	84.	72.	70.	96.	80.	83.	92.	57.	110.	90.	88.9. (Average).
Increase.	27.	32.	27.	29.	22.	22.	32.	17.	26.	28.	23.	16.	16.	25.	22.	21.	22.	14.	28.	20.	23.45. (Average).
Time taken to reach max reading	20.	20.	20.	35.	15.	15.	20.	20.	15.	15.	15.	10.	10.	10.	15.	10.	15.	10.	20.	15.	16.25. (Average).

x. Cases 6, 2, and 4, respectively, of the series of  
six controls quoted below.



control series given below). The readings were taken at 1, 3, 5, 7, 10, 15, 20, 25, 30, and 35 minutes. The time one minute was taken as that point at which the first reading could be observed, i.e. as soon as the acid and blood had been mixed and diluted to a shade fairly equal to the standard. This certainly occupies one minute. The times were gauged with the second hand of a watch - no stop watch being available. The results were as shown on the attached sheet. The average time taken for the maximum colour change to occur was 16.25 minutes, and the average increase of the final over the original reading was 23.45. The average increase which took place in the respective time intervals was:-

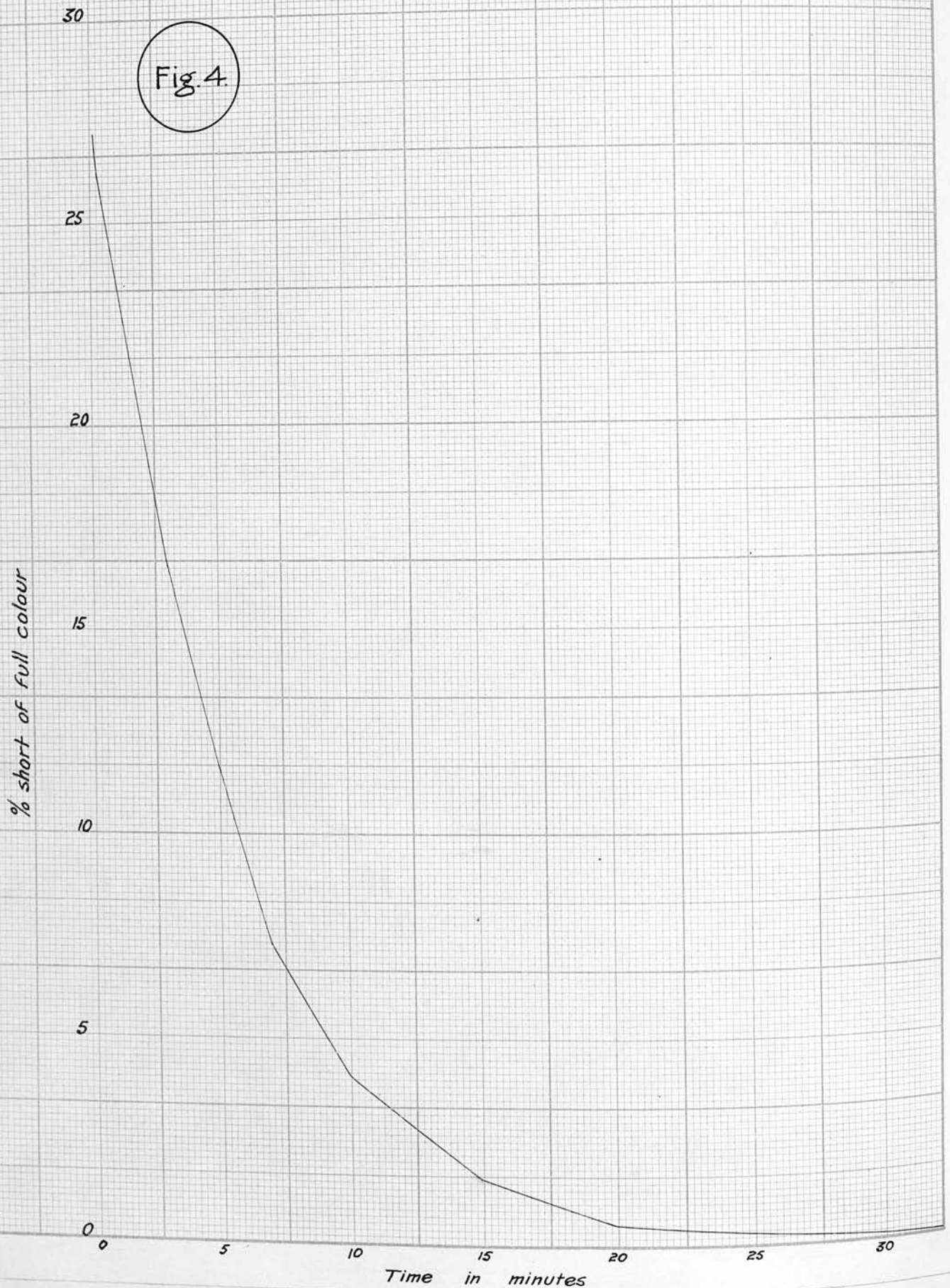
<u>Interval.</u>	<u>Increase.</u>
1 - 3	8.75
3 - 5	4.4
5 - 7	3.85
7 - 10	2.85
10 - 15	2.2
15 - 20	0.95
20 - 25	0.25
25 - 30	0.1
30 - 35	0.1
35 - 40	0.0

The percentage by which the reading at a given time fell short of the final reading was as follows:-

At 1 minute	26.36 %
3 minutes	16.53
5 "	11.58
7 "	7.25
10 "	4.05
15 "	1.57
20 "	0.51.
25 "	0.22
30 "	0.11
35 "	0.00



Graph shewing relationship between depth of colour of acid-haematin and time at which reading is taken.



In studying the spectroscopic characters of a solution of acid haematin with a view to elaborating his well known colorimeter for standardisation of Sahli haemoglobinometers, Newcomber (24) makes a statement to the following effect.

The colour density of a newly prepared solution of acid haematin is expressed by the equation

$$xy = -40$$

where x is the time in minutes and y is the percentage short of full colour. Thus after ten minutes the result is within 4% of the final reading, after twenty minutes within 2%, and after forty minutes the result is within 1% of the final figure.

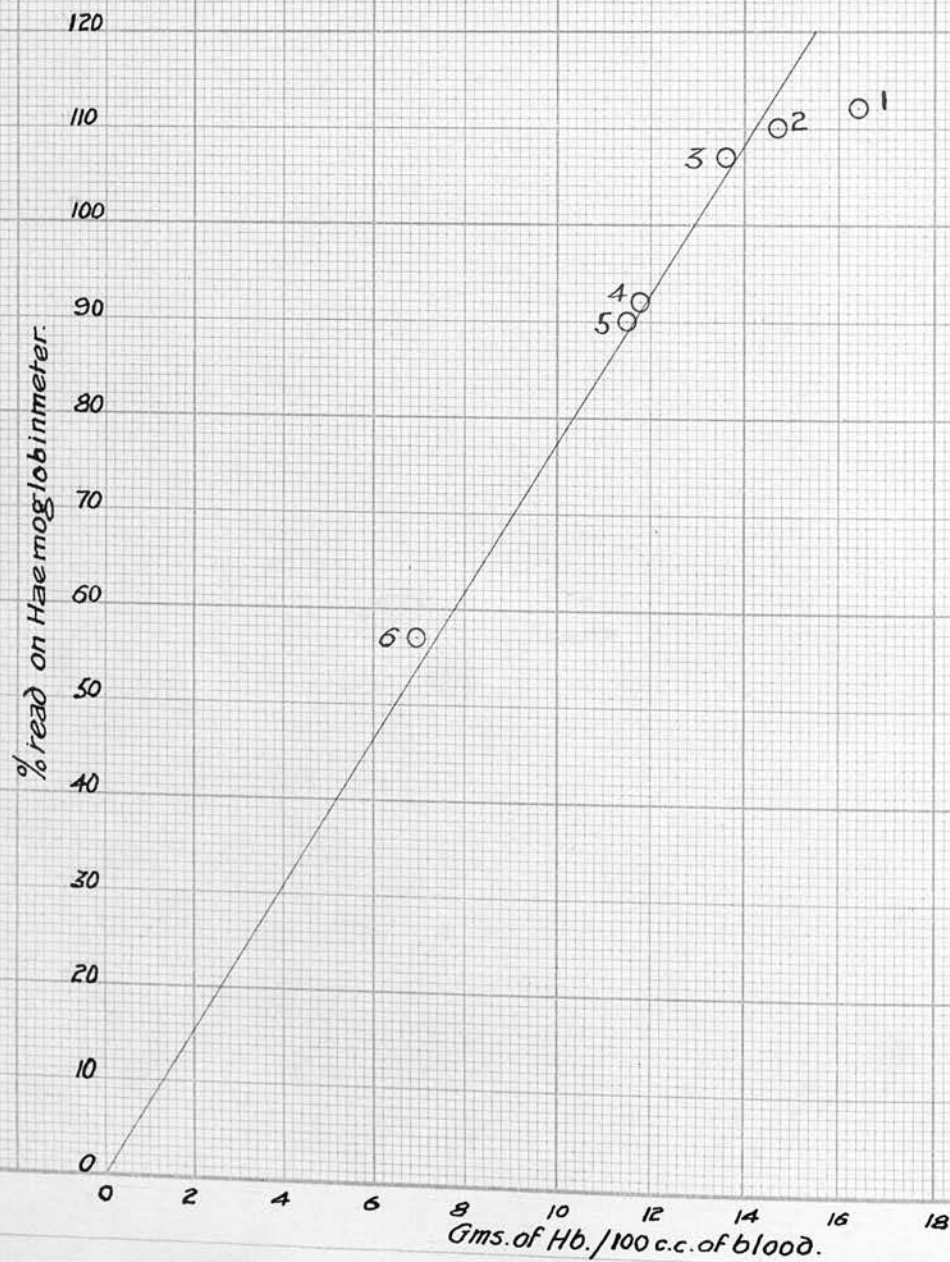
My results show the average reading to be short by 4.05 % at ten minutes, and by 0.51 % at twenty minutes. The results obtained are plotted in a graph and it will be observed that the form of the graph resembles, at least, the rectangular hyperbola which the equation  $xy = -40$  represents. (Fig. 4).

The next step was to standardise my particular instrument against an instrument which was known from first principles to be correctly standardised and have a negligible percentage of error. By the kind permission of Dr C. P. Stewart of the Clinical laboratory of the Royal Infirmary of Edinburgh I was given facilities for standardising my particular instrument in terms of grammes of haemoglobin per 100 c.c. Six specimens of blood were obtained. The percentage of haemoglobin in grammes per 100 c.c. of blood was estimated by means of/

Graph shewing readings on authors instrument in terms of grammes of Hb/100cc. of blood. 6 controlled cases.

Fig 5.

1.	112 = 16.4 Gm.Hb.
2.	110 = 14.7 " "
3.	107 = 13.6 " "
4.	92 = 11.8 " "
5.	90 = 11.5 " "
6.	57 = 6.95 " "

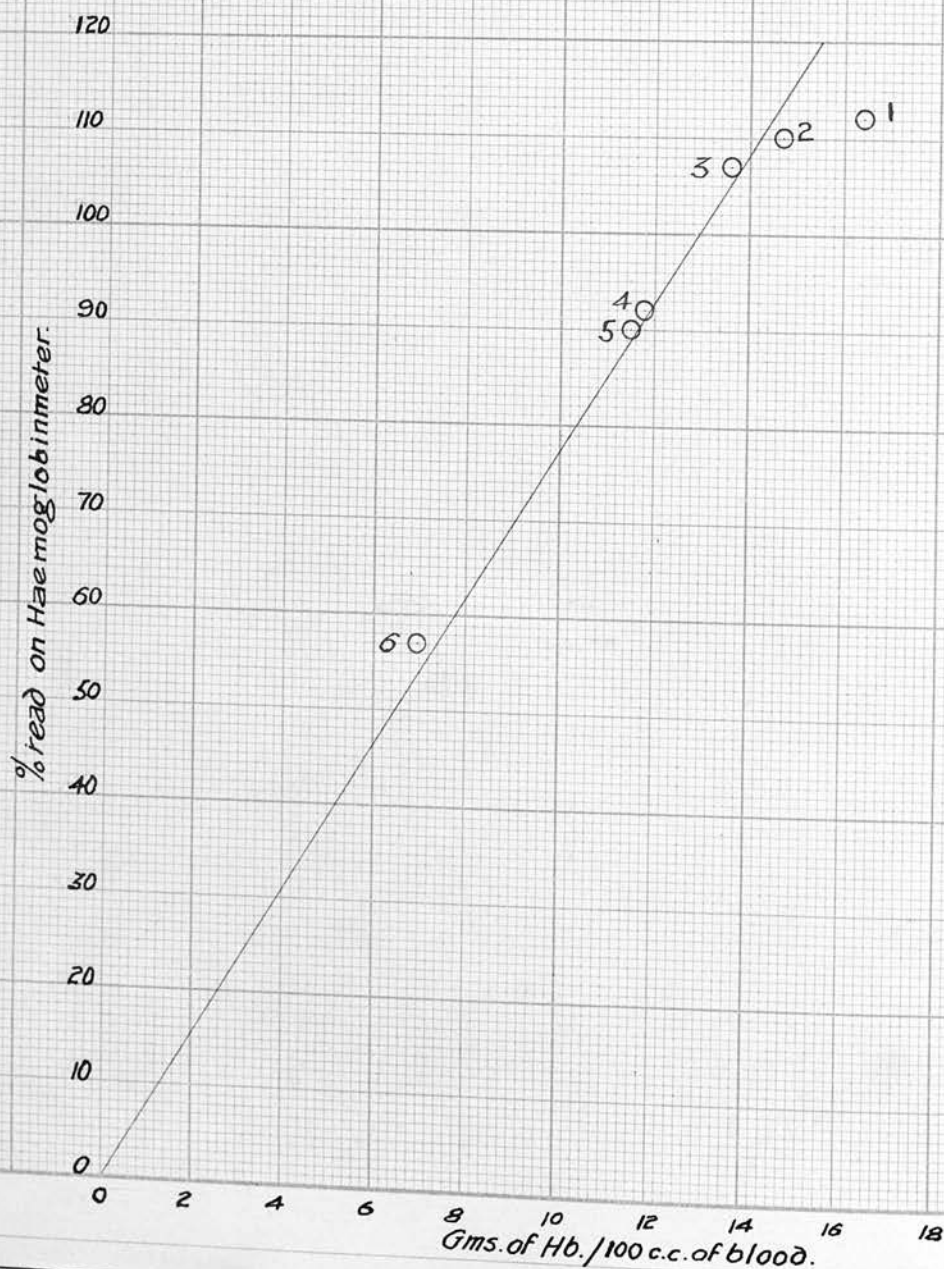




Graph shewing readings on authors instrument in terms of grammes of Hb/100 c.c. of blood. 6 controlled cases.

Fig 5.

1. 112 = 16.4 Gm.Hb.
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5. 90 = 11.5 " "
6. 57 = 6.95 " "





of a Newcomber colorimeter which had been previously checked by the Van Slyke method of estimating oxygen combining power of blood. At the same time I estimated the haemoglobin percentage with my Sahli haemoglobinometer using my own solution and an exactly similar technique to that used in the above series.

The results were:-

Time interval in minutes.	Cases					
	1	2.	3.	4.	5.	6.
1.	88	82	80	70	70	43
3.	97	92	88	76	78	52
5.	101	97	96	82	83	52
7.	106	102	100	86	86	54
10.	109	105	105	88	90	57
15.	112	108	107	90	92	57
20.	112	110	107	90	92	57
25.	112	110	107	90	92	57
Increase.	24	28	27	20	22	14 - average 22.5
Time.	15	20	15	15	15	10 - average 15 minutes.

Haemoglobin  
grammes per  
100 c.c. blood 16.4; 14.7; 13.6; 11.5; 11.8; 6.95  
Newcomber  
colorimeter.

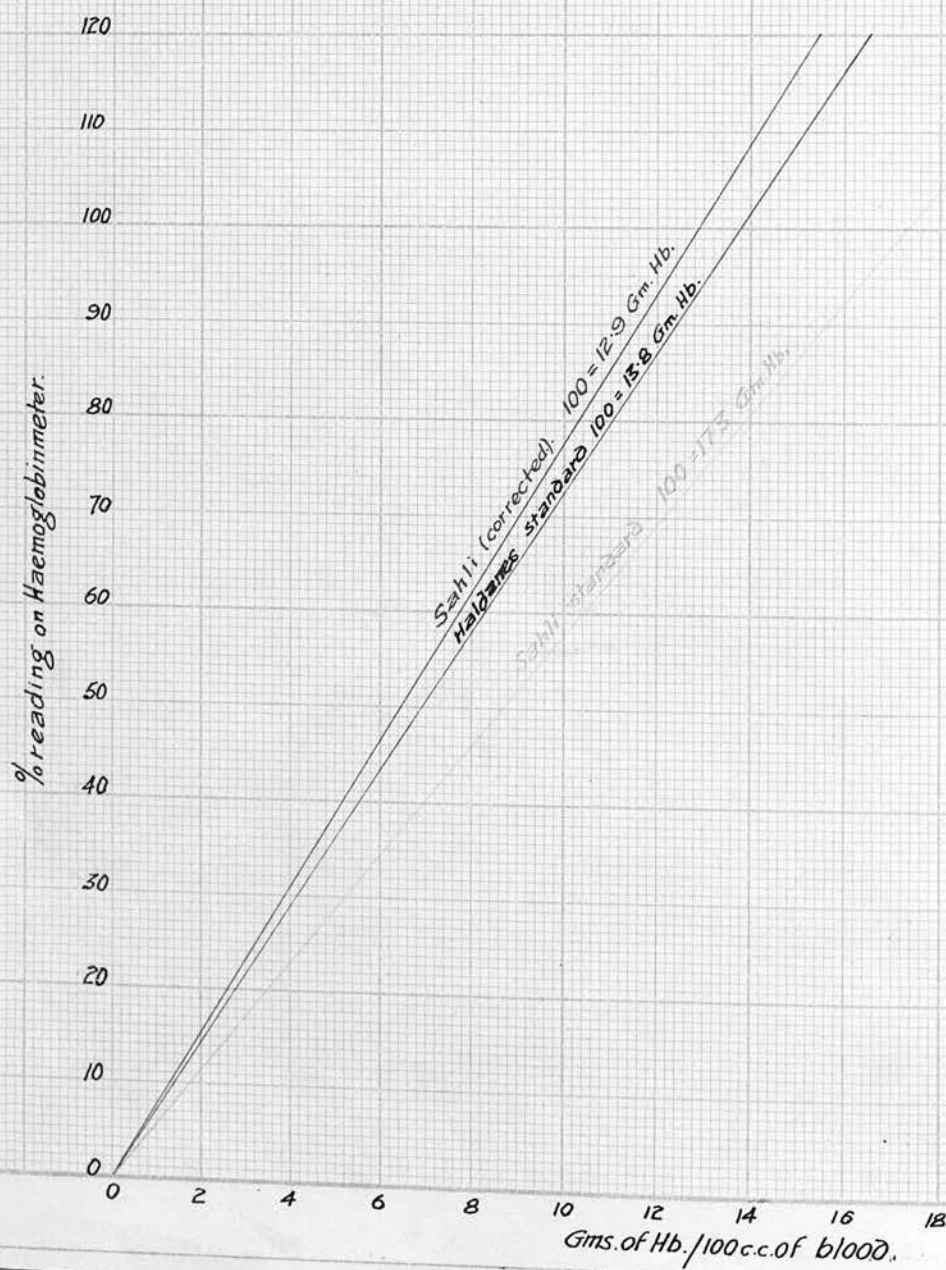
The final readings therefore, are:-

1.	112 %	=	16.4	grammes per 100 c.c. blood.
2.	110	=	14.7	do.
3.	107	=	13.6	do.
4.	90	=	11.5	do.
5.	92	=	11.8	do.
6.	57	=	6.95	do.

(Fig 5) These results are incorporated in a graph as shown. It is observed that apart from the first case, the results can be expressed with reasonable accuracy, in a straight line graph. The first reading was grossly out/

Graph shewing relation between readings on author's instrument and standard Haldanes scale.

Fig. 6.



out of line. I explained this by the fact that my eye had not yet become able to appreciate the "end point" of the colorimeter. The result is nevertheless included for the sake of completeness.

It was possible to construct a graph (Fig. 6) by which the final readings on my instrument could be given in percentages on Haldane's scale.

The abscissa represented grammes of haemoglobin per 100 c.c. of blood, and the ordinate percentage reading on the haemoglobinometer of

(1). Haldane's scale

100 % = 13.8 grammes per 100 c.c. of blood.

(2). Standard Sahli scale

100 % = 17.3 grammes. (25).

(3). My Haemoglobinometer readings at 30 minutes.

100 % = 12.9 grammes.

It will be observed that the results obtained with my instrument were high, that they were uniformly and proportionately so, and that a graph could be constructed enabling the results obtained to be read off in terms of Haldane's standard of 100% = 13.8 grammes per 100 c.c. of blood. Further that the standard for Sahli which the instrument makers, and literature in general informed me was 100% = 17.3 grammes of haemoglobin per 100 c.c., was much too high for my particular instrument.

A fading standard is easily explained, but a non-fade instrument giving such faulty results is upsetting. I can only conclude that the fault lay in the/

the particular piece of glass used or in the opacity of the substance which is used to unite the two glass slabs together.

To return to the original point - namely, at what time interval does the colour of the acid haematin mixture reach a depth which, when matched with that of the standard glass slab, give a reading, which can be directly read off as a percentage on the Haldane scale?

The final readings at 30 minutes were all converted into Haldane percentages by means of the graph (Fig. 6 ). I have arranged these in a vertical column below. Opposite each reading is given the reading on my instrument at 5, 7, and 10 minutes, thus:-

Case.	Final Reading.	Haldane Equivalent.	My readings at		
			5 min.	7 min.	10 min.
1.	90.	84.	77.	82.	86.
2.	95.	89.	81.	85.	88.
3.	98.	92.	85.	92.	94.
4.	112.	104.	90.	93.	96.
5.	90.	84.	82.	85.	87.
6.	90.	84.	81.	87.	89.
7.	97.	91.	85.	87.	92.
8.	63.	58.	55.	57.	58.
9.	101.	95.	87.	92.	94.
10.	108.	97.	97.	99.	103.
11.	84.	78.	74.	76.	80.
12.	72.	67.	68.	70.	72.
13.	70.	65.	64.	66.	70.
14.	96.	89.	85.	94.	96.
15.	80.	75.	72.	76.	78.
16.	83.	77.	78.	80.	83.
17.	92.	85.	80.	86.	90.
18.	57.	54.	52.	54.	57.
19.	110.	103.	97.	102.	105.
20.	90.	84.	82.	86.	88.
Average: 88.9.			82.75.	78.64.	82.49.
				85.34.	

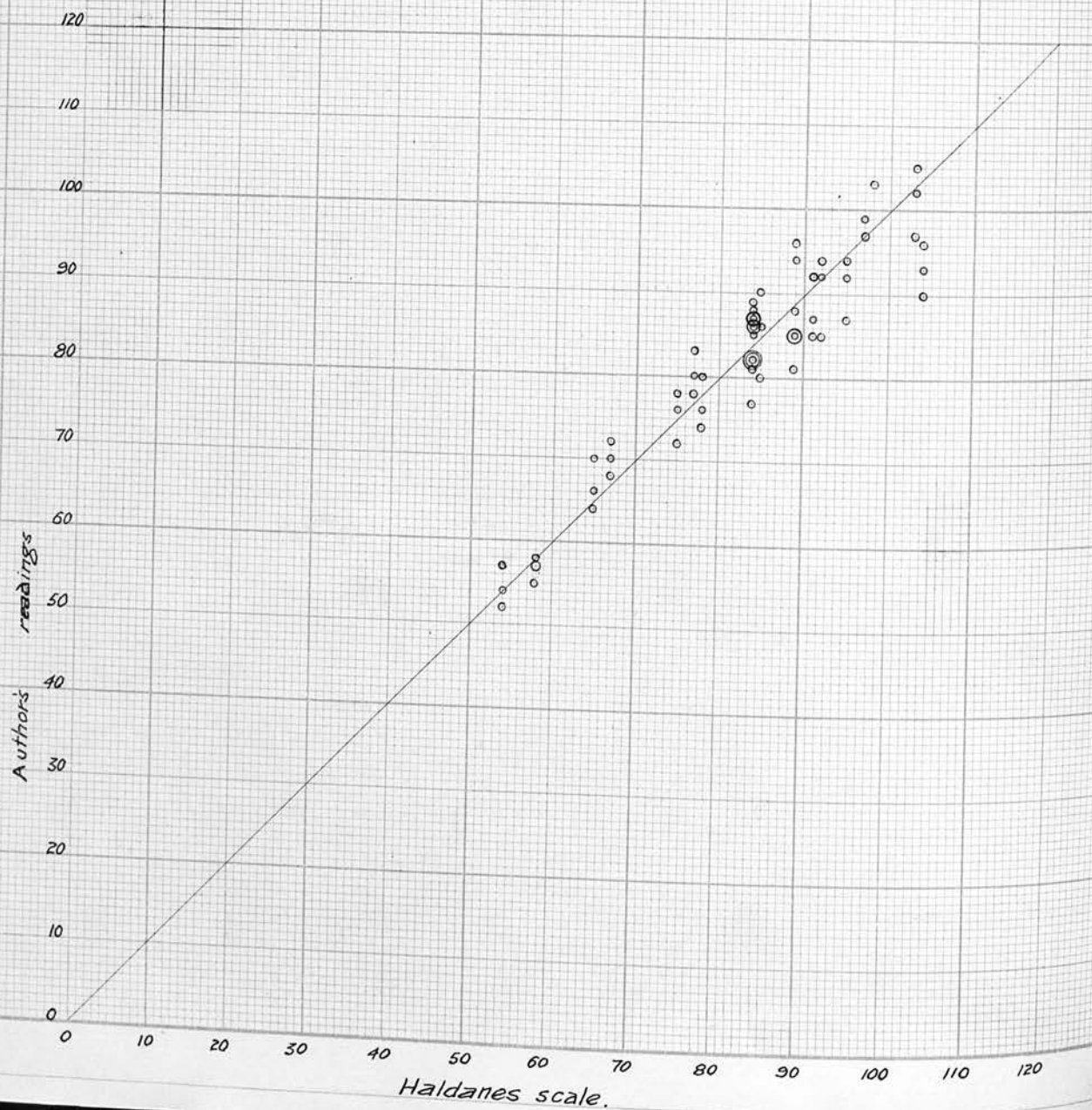
A graph was constructed with percentage readings on/



Graph shewing relation between authors readings at 5, 7, & 10 mins.  
and the corresponding value on the Haldanes scale.

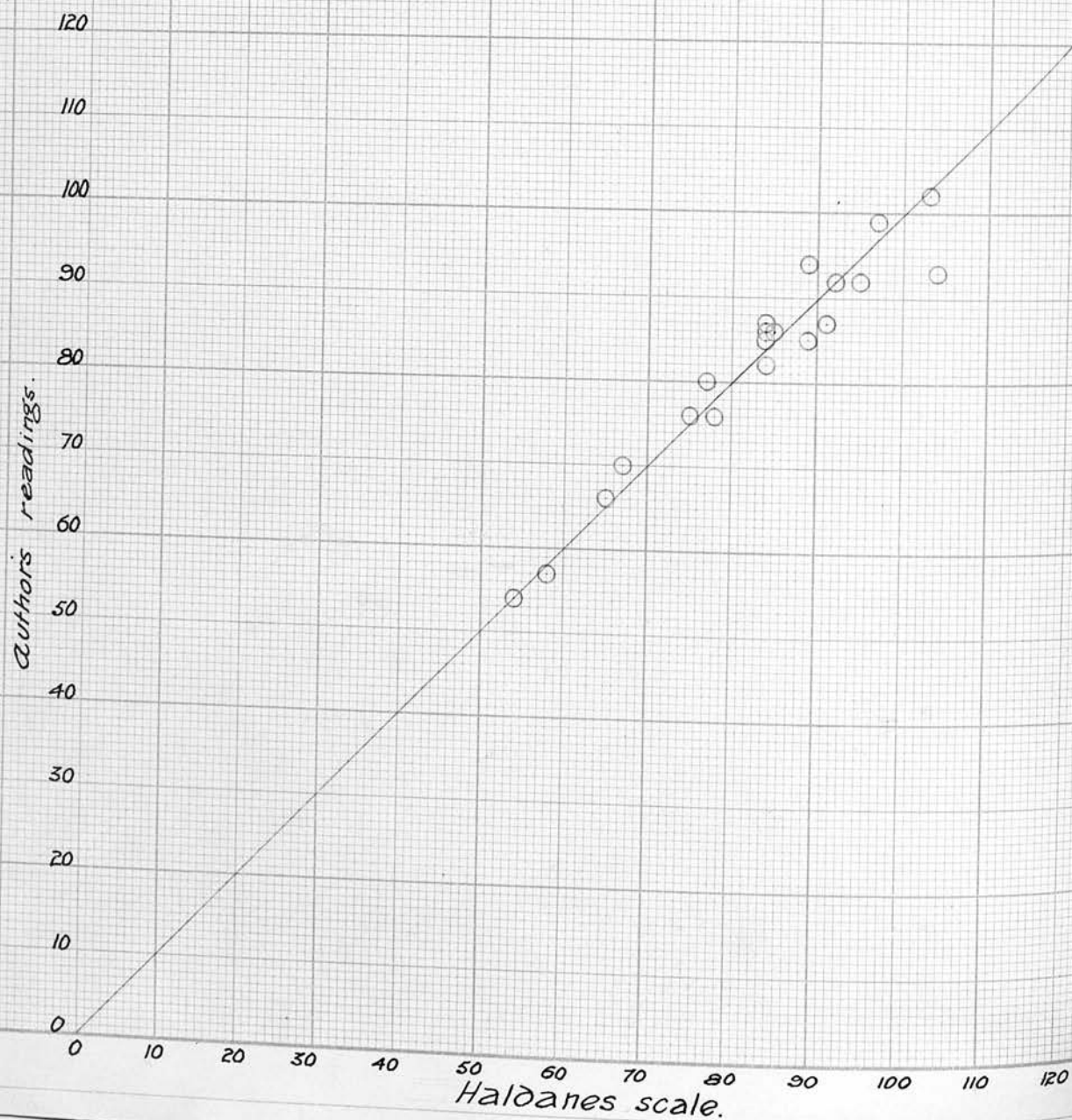
10 minutes in blue  
7 minutes in vermillion.  
5 minutes in brown.

Fig. 7.



Graph shewing relation between authors readings at 7 mins.  
and the corresponding value on the Haldanes scale.

Fig 8.



on the Haldane scale as the abscissa, and my haemoglobinometer readings as the ordinate.

The straight line represents the time interval when 100% on my scale = 100% on the Haldane scale.

A glance at the graph at once reveals the following:- (Fig. 7)

- (1). The readings taken at 5 minutes are uniformly low, i.e. they lie mostly to the right of the straight line.
- (2). The readings at 10 minutes were too high, i.e. they lie mostly to the left of the line.
- (3). The readings at 7 minutes were approximately equal to the Haldane percentages, and all lay on or around the straight line.

A further graph was constructed showing the readings at 7 minutes alone in relation to Haldane percentages. They conform very nearly to a straight line graph.

A further point of considerable significance emerges from a glance at the averages quoted.

The average reading at 30 minutes for the twenty cases was 88.9%. On the graph, Fig. 6 this is shown to represent 82.75% on the Haldane scale.

The average haemoglobinometer reading for the twenty cases at 7 minutes was 82.49% which is short of 82.75 by a gratifyingly negligible amount.

I feel therefore, that I am justified, with the particular/



particular model I use, in taking the readings at 7 minutes as representing actual percentages of haemoglobin on the Haldane scale, with the important provision that the technique I employ must be strictly adhered to, and my definition of what is meant by 7 minutes be clearly understood.

I am now employing this method of obtaining a quick conversion to Haldane percentages in actual practice, but all the figures quoted in this paper are from readings taken at 30 minutes, and converted into Haldane percentages afterwards by means of the graph. Fig.6.

Finally, I should like to draw attention to the fact that no relation could be established between the time taken for the reading to become constant, and the final reading obtained. Talquist (26) who has done a great deal of work on the curves of dissociation of haemoglobin into alkaline haematin, discards the theory that the haemoglobin in the megaloblastic cells of pernicious anaemia is of a different character from normal adult haemoglobin, on the grounds that the dissociation curves of these patients do not vary from those of normal adults.

In the series of twenty cases quoted, two, nos: 2 and 14, were the subjects of pernicious anaemia. One took less time, and one more, than the average, to produce a final constant reading. I had no experience in/



in the use of Sahli's haemoglobinometer in infants, in whom, according to Talquist, the formation of haematin is a very slow progress.

Summary.

- (1). Reasons for suspecting the readings of my instrument are quoted.
- (2). The phenomenon of the gradually increasing colour depth of a newly prepared solution of acid haematin is described.
- (3). Observations have been made on 20 cases and the results obtained are uniform and in agreement with those of the authorities quoted.
- (4). The method of standardising my instrument in terms of grammes of haemoglobin per cent, is described in detail.
- (5). A graph showing the relationship between my final readings and those of the Haldane scale is shown.
- (6). A series of graphs are shown and based on the results obtained from the investigation of the readings of 20 patients at different time intervals.
- (7). It is shown that a reasonably high degree of accuracy can be obtained with the Sahli Haemoglobinometer.
- (8). It is shown that with the particular technique described, and with my particular model the readings taken at 7 minutes correspond/

correspond very closely to the actual  
percentage readings on the Haldane scale.

SMALL VALUE OF AIR FLOOD CORRECTIONS

Normal Values for Red Blood Corpuscles.

The standards of normal for red blood corpuscles vary considerably. I shall confine my remarks to a reference to four publications of normal figures by well-known workers.

1. Price Jones, Vaughan, and Edwards (1917) who

NORMAL VALUE OF RED BLOOD CORPUSCLES.

red cells per cubic millimeter.

2. McDermott (1920) who gives the range 4.85-5.3

3. Hodge and Hynes (1921) whose range was 4.1

- 5.3.

4. Holt and Weinstein (1921) who give the follow-

ing values for children.

2 to 5 years.....4.5 millions.  
10 years.....5.3 do.

Through-out this paper 5,000,000 is accepted as the normal figure from the age of 10 years onwards. In school age from a value of 4,500,000 is considered normal.

Normal Values for Red Blood Corpuscles.

The standards of normal for red blood corpuscles vary considerably. I shall confine my remarks to a reference to four publications of normal figures, by well-known workers.

1. Price Jones, Vaughan, and Goddard ( 27) who give the normal range of 4.53 - 6.31 million red cells per cubic millimetre.
2. McGeorge ( 28) who gives the range 4.85-6.37.
3. Whitby and Hynes ( 29) whose range was 5.1 - 6.5.
4. Holt and McIntosh (30 ) who give the following values for children.

2 - 5 years.....4.5 millions.  
10 years.....5.0 do.

Through-out this paper 5.000.000. is accepted as the normal figure from the age of 10 years onwards. In school children a value of 4.500.000. is considered normal.



## The Micrometer.

Perhaps the greatest authority on the measurement of red cell diameter is O. Price Jones, whose curves of mean cell diameters are known to all hematologists.

### THE MEAN CELL DIAMETER.

its estimation by a diffraction method. with variations of 6.634  $\mu$ . to 7.634  $\mu$ .

Most workers take 7.5  $\mu$ . as a fair average for the mean red cell diameter.

The only certainly accurate method of arriving at the mean cell diameter is by using the Price Jones technique (20) but other methods are in use at the present day.

I refer chiefly to the diffraction method. The principle underlying this method is that a halo is formed by the diffraction of rays of light from a constant source, then they pass through a glass slide on which is a film of blood. The size of the halo is indirectly proportional to the size of the fixed corpuscles causing the diffraction. The method is only accurate when the cells are spread on a film so as to touch each other tangentially without overlapping. To prepare such a film is a task which is well-nigh impossible.

The micrometer gives, of course, an average diameter, and does not take into consideration the presence of any degree of anisocytosis.

The Halometer.

Perhaps the greatest authority on the measurement of red cell diameter is C. Price Jones, whose curves of mean cell diameters are known to all haematologists.

In a recent publication (31) he gives the mean diameter of red blood corpuscles as  $7.17 \pm 0.017$  u. with variations of 6.654 u. to 7.686 u.

Most workers take 7.2 u. as a fair average for the mean red cell diameter.

The only certainly accurate method of arriving at the mean cell diameter is by using the Price Jones technique (32) but other methods are in use at the present day.

I refer chiefly to the diffraction method. The principle underlying this method is that a halo is formed by the diffraction of rays of light from a constant source, when they pass through a glass slide on which is a film of blood. The size of the halo is indirectly proportional to the size of the blood corpuscles causing the diffraction. The method is only accurate when the cells are spread on a film so as to touch each other tangentially without overlapping. To prepare such a film is a feat which is well-nigh impossible.

The halometer gives, of course, an average diameter, and does not take into consideration the presence of any degree of anisocytosis.

Fullerton/

Fullerton, Lyall and Davidson (33 ) have investigated this subject. They state that the chief sources of error in the use of this instrument are the following:-

- (1). Films spread too slowly causing undue separation of the cells, especially if anaemia is present.
- (2). Films spread too quickly causing overlapping and a consequently wrong impression of megalocytosis.
- (3). The presence of anisocytosis or poikilocytosis causing blurring.
- (4). The personal factor in the appreciation of the colour of the halo.

They hold very strong views on the subject and state,

"The chief objection to the diffraction methods is that they suggest to the general practitioner that he has at his disposal a means by which he can make a simple, rapid, and accurate, diagnosis of the type of the anaemia present without the assistance of a full haematological examination. We are of the opinion that such a conclusion is not only unjustified, but may be a source of danger to the patient."

Below I append the results of 116 readings with Allenbury's Halometer and accompanying each reading is the colour index obtained by estimating the Haemoglobin and R.B.C. percentage at the time at which the halometer reading was made. For reference the cases/

cases on whom the observations were made are quoted. I have arranged the readings in order, according to the colour indices - the highest colour index, 1.46, at the top, and the lowest, 0.54, at the bottom.

I hoped to see the halometer readings become gradually less as the colour index became lower. There was, however, no such parallel change.

The only point I made out was that in the cases of pernicious anaemia (cases 83, 84, 85, 86) the halometer reading was always 7.4 or above that figure. The highest colour index (1.46) was obtained in the patient with the highest halometer reading (8.1 u.) but apart from this, no definite correspondence existed between the colour index and the halometer reading.

I therefore concluded that, although I obtained uniformly high halometer readings in cases of pernicious anaemia, on the whole, the instrument used did not give even an approximate idea of the mean cell diameter.

As Fullerton points out, the personal factor is an ominously large one, and the high readings in the patients with pernicious anaemia may have been a case of the 'wish being father to the thought.'

Booth (34) describes an exceedingly crude method of obtaining an idea of the degree of megalocytosis/

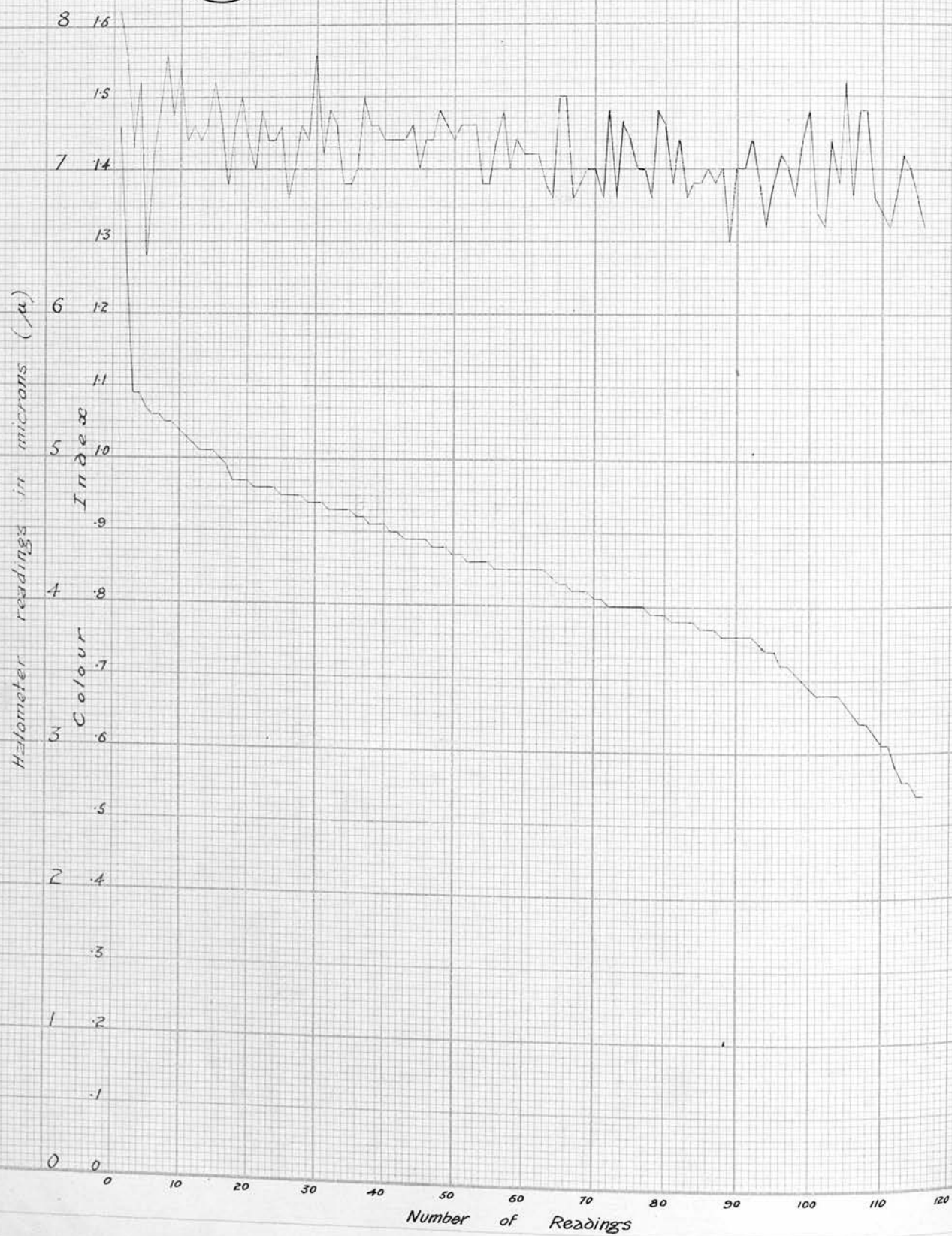


megalocytosis in a suspected case of pernicious anaemia.

The observer prepares two blood films, one of normal blood and one of the suspected case of pernicious anaemia. He gazes through them both, simultaneously, at a fixed source of light. By the aid of external pressure on the eyeball a diplopia is produced, and so the two haloes can be compared. By changing the film the element of suggestion can be eliminated.

Graph shewing Halometer reading given in particular case of  
Colour Index shewn below. 116 observations.

Fig 9.



<u>Case No:</u>	<u>Colour Index.</u>	<u>Halometer Reading.</u>
85.	1.46.	8.1.
84.	1.33.	7.8.
74.	1.09.	7.2.
86.	1.09.	7.6.
2.	1.07.	6.4.
3.	1.06.	7.1.
66.	1.06.	7.4.
83.	1.05.	7.8.
85.	1.05.	7.4.
85.	1.04.	7.7.
61.	1.03.	7.2.
62.	1.02.	7.3.
69.	1.01.	7.2.
81.	1.01.	7.3.
50.	1.01.	7.6.
41.	1.00.	7.3.
19.	0.99.	6.9.
76.	0.97.	7.3.
35.	0.97.	7.5.
35.	0.97.	7.2.
18.	0.96.	7.0.
48.	0.96.	7.4.
73.	0.96.	7.2.
8.	0.96.	7.2.
53.	0.95.	7.3.
36.	0.95.	6.8.
41.	0.95.	7.0.
41.	0.95.	7.3.
51.	0.94.	7.2.
72.	0.94.	7.1.
1.	0.94.	7.8.
42.	0.93.	6.9.
75.	0.93.	7.4.
81.	0.93.	7.3.
16.	0.92.	7.0.
14.	0.92.	7.5.
43.	0.99.	7.3.
75.	0.91.	7.3.
81.	0.91.	7.2.
59.	0.90.	7.2.
44.	0.90.	7.2.
52.	0.89.	7.2.
64.	0.89.	7.3.
78.	0.89.	7.0.
1.	0.89.	7.2.
28.	0.88.	7.2.
85.	0.88.	7.4.
33.	0.88.	7.3.
21.	0.87.	7.2.
74.	0.87.	7.3.
9.	0.86.	7.3.
21.	0.86.	7.3.
24.	0.86.	6.9.
26.	0.86.	6.9.

<u>Case No.:</u>	<u>Colour Index.</u>	<u>Halometer Reading.</u>
24.	0.85.	7.2.
46.	0.85.	7.4.
52.	0.85.	7.0.
54.	0.85.	7.2.
57.	0.85.	7.1.
63.	0.85.	7.1.
81.	0.85.	7.1.
74.	0.85.	6.9.
58.	0.84.	6.8.
4.	0.83.	7.5.
15.	0.83.	7.5.
13.	0.82.	6.8.
18.	0.82.	6.9.
28.	0.82.	7.0.
41.	0.81.	7.0.
60.	0.81.	6.8.
4.	0.82.	7.4.
17.	0.80.	6.8.
17.	0.80.	7.3.
31.	0.80.	7.2.
34.	0.80.	7.0.
22.	0.80.	7.0.
22.	0.79.	6.8.
32.	0.79.	7.4.
33.	0.79.	7.3.
44.	0.78.	6.9.
68.	0.78.	7.2.
51.	0.78.	6.8.
79.	0.78.	6.9.
20.	0.77.	6.9.
30.	0.77.	7.0.
47.	0.77.	6.9.
49.	0.76.	7.0.
55.	0.76.	6.5.
57.	0.76.	7.0.
40.	0.76.	7.0.
17.	0.76.	7.2.
12.	0.75.	6.9.
15.	0.74.	6.6.
51.	0.74.	6.9.
70.	0.72.	7.1.
80.	0.72.	7.0.
29.	0.71.	6.8.
66.	0.70.	7.2.
8.	0.69.	7.4.
37.	0.68.	6.7.
60.	0.68.	6.6.
74.	0.68.	7.2.
78.	0.68.	6.9.
11.	0.67.	7.6.



<u>Case No:</u>	<u>Colour Index.</u>	<u>Halometer Reading.</u>
45.	0.65.	6.8.
8.	0.64.	7.4.
14.	0.64.	7.4.
9.	0.63.	6.8.
39.	0.61.	6.7.
71.	0.61.	6.6.
31.	0.58.	6.8.
10.	0.56.	7.1.
27.	0.56.	7.0.
56.	0.54.	6.8.
60.	0.54.	6.6.

### Conclusion.

From a survey of the figures given above and a study of the graph Fig. 9 which has been prepared from them, I conclude that the halometer I used is in no way more accurate than the primitive method described by Booth.

# ANAEMIA IN CHILDREN

Fourteen out of the 17 children were examined. These are, cases 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, and 14.

An interesting table is appended to the report of 14 of these cases. 14 CASES OF ANAEMIA IN CHILDREN.

The cases are in the order in which they were first seen, as follows:

1. A. J. ...	2. ...
3. ...	4. ...
5. ...	6. ...
7. ...	8. ...
9. ...	10. ...
11. ...	12. ...
13. ...	14. ...

The following table shows the results of the examination of the 14 cases. It is arranged in the order in which the cases were first seen. The table is arranged in the following order: Name, Age, Sex, Date of admission, Date of discharge, Duration of illness, Cause of illness, Treatment, Result.

The results of the examination of the 14 cases are as follows:

Case 1. A. J. ...

Case 2. ...

Case 3. ...

Case 4. ...

Case 5. ...

Case 6. ...

Case 7. ...

Case 8. ...

Case 9. ...

Case 10. ...

Case 11. ...

Case 12. ...

Case 13. ...

Case 14. ...

### Anaemia in Children.

Fourteen out of the 17 children were anaemic. These are, cases 1, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 14, 15, and 17.

An underlying toxic condition was found in 11 of these cases, leaving 3 cases only where no organic lesion was demonstrated.

The cases with an organic background fall into 7 groups, as follows:-

		<u>Case No.:</u>
Tuberculosis.	2 cases.	7, 11.
Threadworms.	2 do.	4, 6.
'Rheumatism.'	2 do.	14, 17.
Diphtheria.	1 case.	10.
Whooping-cough.	1 do.	5.
Bronchitis.	1 do.	3.
Oral sepsis.	2 cases.	9, 12.

---

Total.                      11 cases.

The remaining 3 cases are nos: 1, 8, and 15.

Case 1.      was a little girl of three, who was only mildly anaemic. (Haemoglobin 69). She nevertheless responded very well to Ferri. et. ammon. cit. gr. x, t.i.d.

Case 8.      was a little boy aged 7. It was not even possible to exclude the element of sepsis in his case as he had two carious teeth. He responded very well to Ferri. et. ammon. cit. gr. xx, ti.d.

Case 15.     was a girl of 10 from a good class home.

No cause of her anaemia could be found. The blood/

blood picture did not improve with Syrup Minadex but showed rapid improvement when Ferrous sulphate gr. iii, t.i.d. was exhibited.

#### Response to Treatment.

4 cases were given Ferri. et. ammon. cit. and showed a marked improvement. 1 case failed to improve (no. 17) and, as he did not report, was not seen again until one year later when Ferrous sulphate was prescribed. 10 cases were given Ferrous sulphate and with only one exception, improved at once. Case 7 was an insidiously developing Tabes mesenterica who was given Ferrous sulphate but did not improve. Two cases, 14 and 15, did not show improvement with Syrup Minadex, 3 i, t.i.d. Conclusion - The treatment of anaemia in children with the exhibition of medicinal iron and attention to the underlying cause gives uniformly good results. The Ferrous sulphate gr. iii, tablet is an easily taken and thoroughly reliable preparation.

With regard to the incidence of anaemia in childhood Leonard Findlay has recently published his results on the investigation of 290 patients of the hospital class in the East End of London. He states (35).

"Anaemia in later infancy and childhood is rare, and when present is probably the result of toxic conditions."

Helen/



100.

Helen M. M. McKay (36 ) finds it difficult to accept these results and objects to:-

- (1). Findlay's lower limit of normal, and
- (2). his method of collecting blood by puncturing the lobe of the ear.

The views of Davidson and Fullerton ( 37 ) are more in keeping with those of Leonard Findlay. They state that in 102 children between 2 and 11 years of age:-

"12% of the 3 year old group were this level (70% Haldane scale) and from 5 years onwards all the children had Haemoglobin levels greater than 80%."

From my own observation I draw the following conclusions.

- (1). Anaemia, for which there is no apparent cause, is rare in children.
- (2). Anaemia is a fairly common concomitant syndrome in children unwell from other causes.
- (3). Anaemia in children nearly always responds quickly and dramatically to the exhibition of a suitable iron preparation and attention to the underlying cause.
- (4). An anaemia not responding to treatment, in a child should receive the most thorough investigation.
- (5). The possibility of a tuberculous focus should always be borne in mind. A strikingly/

strikingly high incidence of tuberculosis in anaemic children was found (it may be a fortuitous one). Two cases, (nos: 7 and 11) with a possible third (no: 17) occurred in a series of 14 cases of anaemia in children.

1 CASE OF ANAEMIA IN ADOLESCENCE

I examined two children of the adolescent period. The first was a boy of 15 (case 19) I suspected to be suffering from lead poisoning. He was not, however, anemic. A cursory glance at the blood picture suggested that he might be.

1 CASE OF ANAEMIA IN ADOLESCENCE.

The other case was a girl of 16, who showed a fairly marked fall in hemoglobin (4.57 g/100) and a colour index of 0.81, therefore, not markedly reduced.

She had been the subject of repeated attacks of weakness and had recovered from such an attack only a few weeks before I examined her.

I am inclined to consider her as an anemic rather than a subject of infection or other toxic condition.

The finding of only one case of anemia in a survey of 86 patients of all ages is very striking in view of the fact that anemia is a common condition less than one percent of the population.

This suggests chlorosis as a hematological condition and could find in 1931 (35) that a survey of 113 to 120 cases of anemia in the preceding 2 years.

I examined two children of the adolescent period. One, a boy of 16 (case 19) I suspected to be suffering from lead poisoning. He was not, however, anaemic though a cursory glance at the mucous membranes suggested that he might be.

The other case was a girl of 16, who showed a mild degree of anaemia (Haemoglobin 75) with a fairly normal red cell level (4.670.000) and a colour index which was, therefore, not markedly reduced.

She had been the subject of repeated attacks of bronchitis and had recovered from such an attack only a few weeks before I examined her.

I am inclined to consider her as an anaemia following upon an acute infection rather than a purely idiopathic form.

The finding of only one case of anaemia of adolescents in a survey of 86 patients of all ages is very striking in view of the fact that chlorosis was such a common condition less than one generation ago.

Witts regards chlorosis as a haematological curiosity and could find in 1931 (38) only 5 cases, in a comprehensive survey of all cases of anaemia admitted to Guy's Hospital in the preceding 9 years.



### Pernicious Anaemia.

Seven cases of Pernicious Anaemia were observed. Four were in the series in general practice, cases 53, 54, 55, and 56; the remaining three were observed when I was a Resident House Physician. They had been treated with Camphor, and I changed to Arsenic in each case as part of an investigation into the relative potency of

### 7 CASES OF PERNICIOUS ANAEMIA.

I propose to confine my observations in this section to two points, viz.

- (1). The blood pressure in pernicious anaemia
- and (2). the results of 'depot' therapy.

#### Blood Pressure.

Davidson and Gillies (32) in their excellent textbook on pernicious anaemia state,

"The arteries are usually soft and the blood pressure low."

They mention, however, two cases with systolic pressures of 180 and 230.

In the British Medical Journal of last year I read two articles by H. S. McGregor (33) and John W. Milkinson (34) respectively, on the association of diabetes mellitus and pernicious anaemia. Neither of these writers indicated the blood pressure in the particular cases quoted, but in McGregor's article/

### Pernicious Anaemia.

Seven cases of Pernicious Anaemia were observed. Four were in the series in general practice, cases 83, 84, 85, and 86; the remaining three were observed when I was a resident House Physician. They had been treated with Campolon, and I changed to Anahaemin in each one as part of an investigation into the relative potency of these two liver extracts.

I propose to confine my observations in this section to two points, viz.

(1). The blood pressure in pernicious anaemia and (2). the results of 'depot' therapy.

### Blood Pressure.

Davidson and Gulland (39) in their excellent textbook on pernicious anaemia state,

"The arteries are usually soft and the blood pressure low."

They mention, however, two cases with systolic pressures of 180 and 220.

In the British Medical Journal of last year I read two articles by H. G. McGregor (40) and John F. Wilkinson (41) respectively, on the association of diabetes mellitus and pernicious anaemia. Neither of these writers indicated the blood pressure in the particular cases quoted, but in an earlier article/

article J. F. Wilkinson (42 ) reviewed the whole question of the association of other diseases with pernicious anaemia.

This authority made a careful study of 370 cases of pernicious anaemia of whom 98 had co-existing disease. Of these, 8 patients (2.16% of the whole series) had hyperpiesis. Their systolic blood pressures ranged from 150 to 240 m.m. of mercury.

The author ignores any possible connection between hypertension and pernicious anaemia. He suggests that achlorhydria was the most striking common factor in pernicious anaemia and the concomitant conditions found in the 98 cases quoted.

In a study of the relationship between the viscosity of the blood and the existing blood pressure D. M. Lyon made the following statement(43)

"In health a close parallelism can be demonstrated to exist between the increase of blood pressure and the blood viscosity up to middle life, but after this the agreement ceases."

The second point of this statement is a very important qualification of his further finding that

"in a patient with anaemia or cyanosis the alteration in the B.P. is very largely dependent on the thinness and thickness of the blood since the normal mechanism of compensation cannot be brought into action."

He comes to the logical conclusion that the normal mechanism - vaso constriction - would further endanger/

endanger the already precarious food supply of the tissues.

In two of my cases, 84 and 85, hypertension was known to exist before the development of pernicious anaemia. I am fortunate in the fact that my principal has written a thesis on essential hypertension and that these cases, 84 and 85 have been under observation by him. He was able to tell me, from his notes, that their blood pressures 10 years ago were 260/140 and 240/110 respectively. This is an interval of 9 years and 4 years respectively, before pernicious anaemia was diagnosed. I suggest that the long continued hypertension, with its consequent vaso constriction and medial hypertrophy has resulted in an impoverished blood supply to the gastric mucous membrane.

We can conceive, at least, that such a state might,

(1). interfere with the elaboration of the intrinsic (haemopoietin) factor of the "anti-anaemia complex,"

or (2). interfere with the absorption of the extrinsic factor,

or (3). possibly an element of both was present.

If this theory is accepted, and it is perhaps going too far to elaborate a theory on the observation of only two cases, then we can conclude that nature, from/



from a choice of many evils, accepted what seemed to her the least. A raised blood pressure, although it endangered the blood supply of the stomach, was strongly indicated, in order to provide with blood, what to her mind were more important structures, namely the kidneys and brain.

#### Depot Treatment of Pernicious Anaemia.

The treatment of pernicious anaemia by intramuscular injection of liver extract is a comparatively recent procedure.

J.G. McCrie ( 44) gives an excellent summary of the march of events which lead to the exhibition of liver in the treatment of pernicious anaemia. He sums up all the literature which was available in 1928. It was only in 1931 that an injectable liver extract was announced by Gansslen. (45 ).

The earlier parenteral extracts were not quite so potent as our present day fractions, and transfusion remained the sine qua non in all cases of moderate severity as a commencement to treatment. Thus in 1933 J.D. Comrie (46 ) quotes a case where transfusion had to be resorted to before the intravenous liver extract was effective.

As the technique of manufacture of liver extract improved the conception of the 'depot' treatment of liver therapy emerged.

Goodall (47 ) described his results in the treatment/

treatment of several cases by injection at monthly intervals of pernaeman forte. He found the average dose to be about 5 c.c.

In 1936 R.M. Murray-Lyon (48 ) described in detail his results in

"a case of successful maintenance in pernicious anaemia by the depot treatment after seven years' oral therapy had proved unsatisfactory for maintenance."

He also referred to 7 other successful cases.

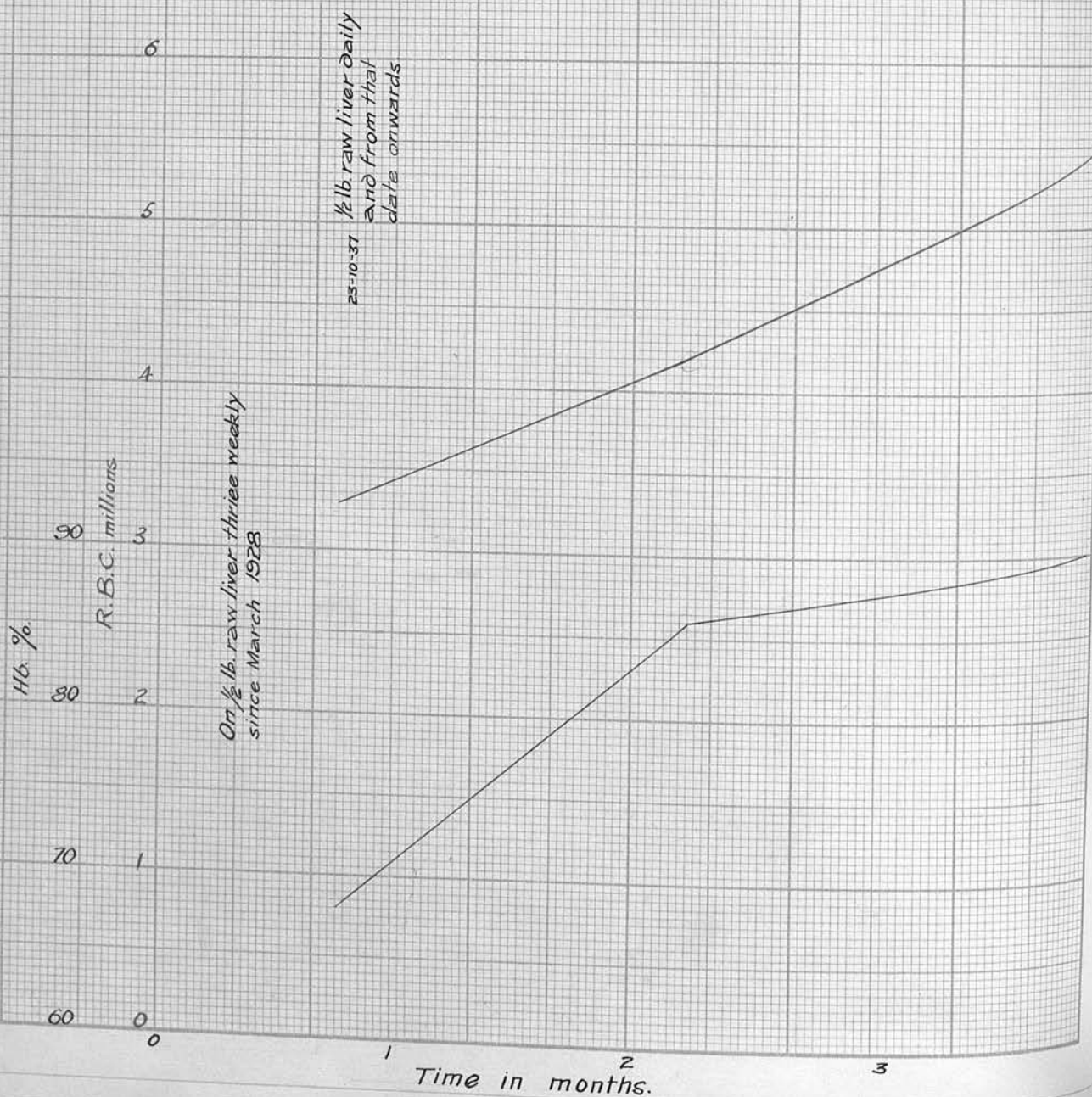
A marked advance was made in 1935 by Dakin and West (49 ) who published the results of their experiments on the concentration of liver extract. They were investigating the nature of the formula for the "anti anaemic complex." Their extract was not, however, of the high degree of potency expected. The following year they published further investigations (50 ) into the chemical nature of the haemopoietic substance in liver and the fraction, which was named Anahaemin, was infinitely more potent.

This substance was tried out clinically under the auspices of the Medical Research Council by Ungley, Davidson, and Wayne, who studied its effect on 36 cases of pernicious anaemia. (51) They obtained uniformly good results and were of the opinion that the extract was as good as any other known in the treatment of cases with neurological manifestations.

Ungley alone published (52 ) a further series of 20 cases, treated with the more concentrated 'anahaemin' liver extract. Using the double-reticulocyte response/

Graph shewing progress notes of case N° 83 shewing  
blood counts and treatment given.

Fig. 10





response method of Minot and Castle (53) he found the new extract to be

"not less than  $2\frac{1}{2}$  times as potent as the original anahaemin."

The following year Janet Vaughan said that (54)

"personally if she suffered from pernicious anaemia nothing would induce her to be treated with anahaemin. She would prefer less pure preparations. She also calculated that it would have cost her £7 to cure her last patient with anahaemin."

This statement aroused many objections and Dr Vaughan later retracted both statements. She pointed out that,

- (a). her objection to anahaemin had been purely theoretical, and
- (b). her calculation of £7 was based on an out of date price.

Most other writers are unanimous in their praise of anahaemin. Davidson and Fullerton (55), Ungley (56), and Page and Wood (57) are all enthusiastic about its potentialities.

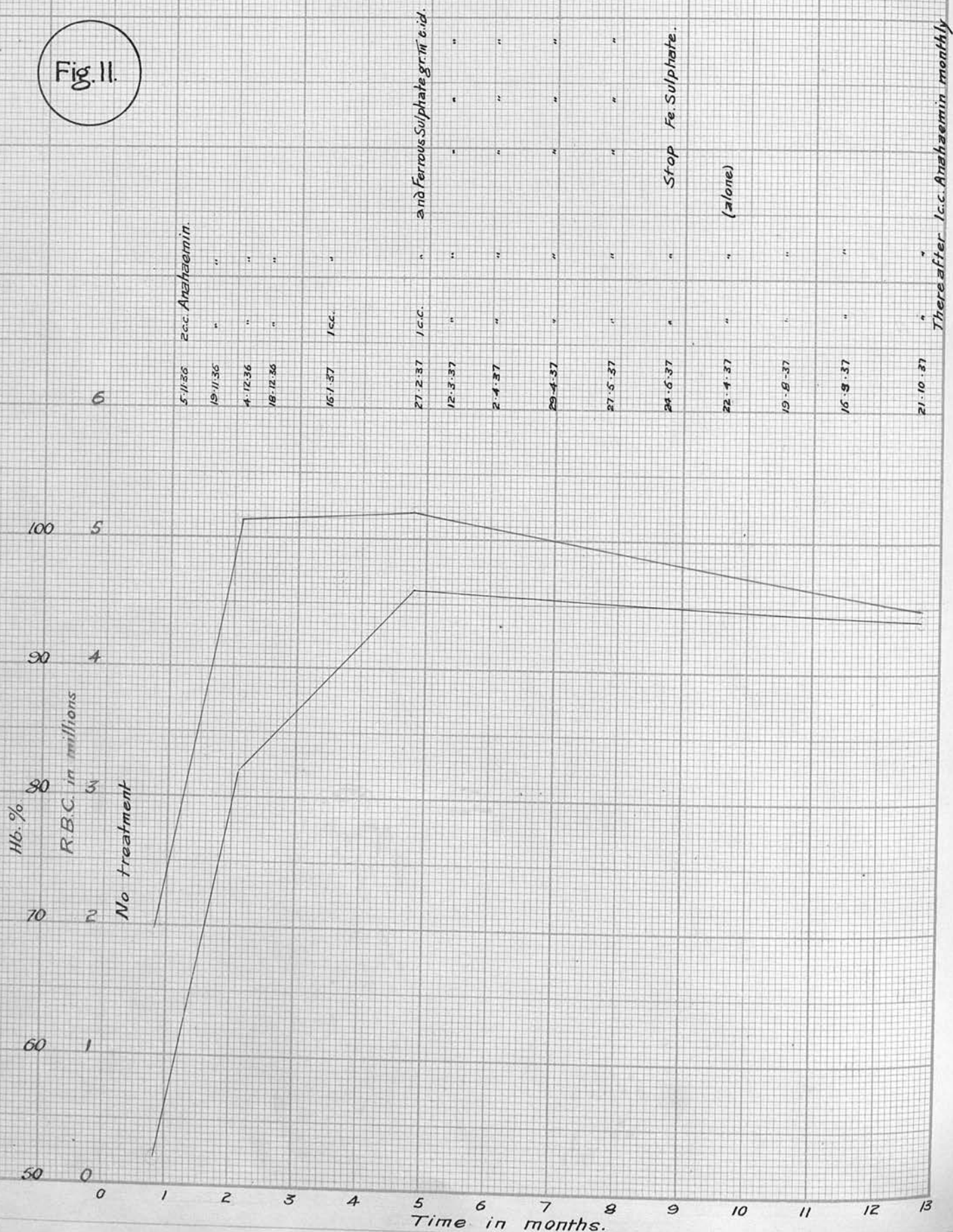
I shall briefly recapitulate the notes on the treatment of my seven cases. Four cases are described which were treated in general practice. They are:-

Case 83: Was diagnosed in March 1928 and has been taking raw ox liver since that time. I first saw him in October, 1937. At the end of January 1938 his blood figures were excellent and he had no therapy other than raw liver orally./



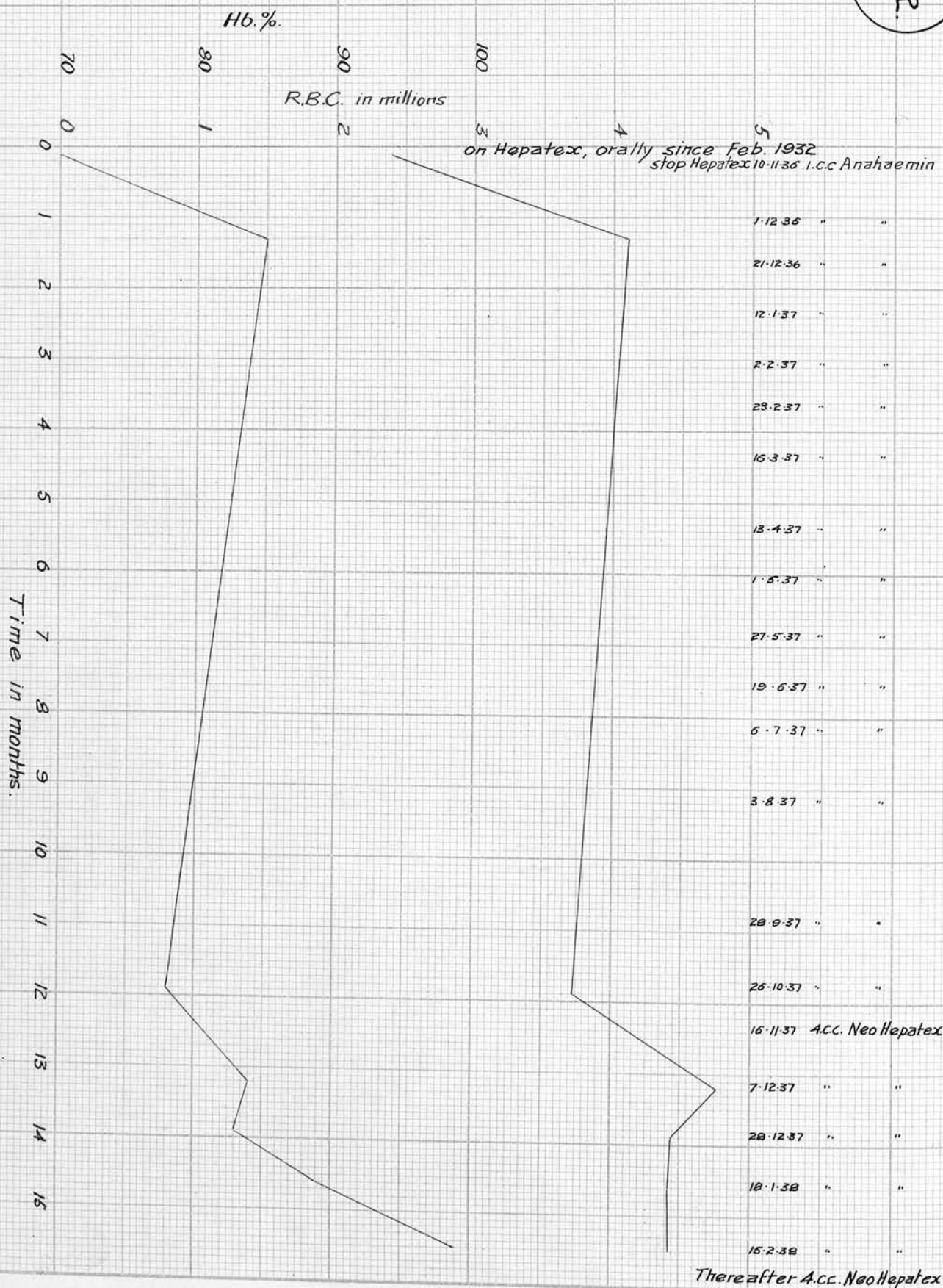
Graph shewing progress notes of case N°84 shewing blood counts and treatment given.

Fig. II.



Graph showing progress notes of case N°85 showing blood counts and treatment given.

Fig. 12.





orally.

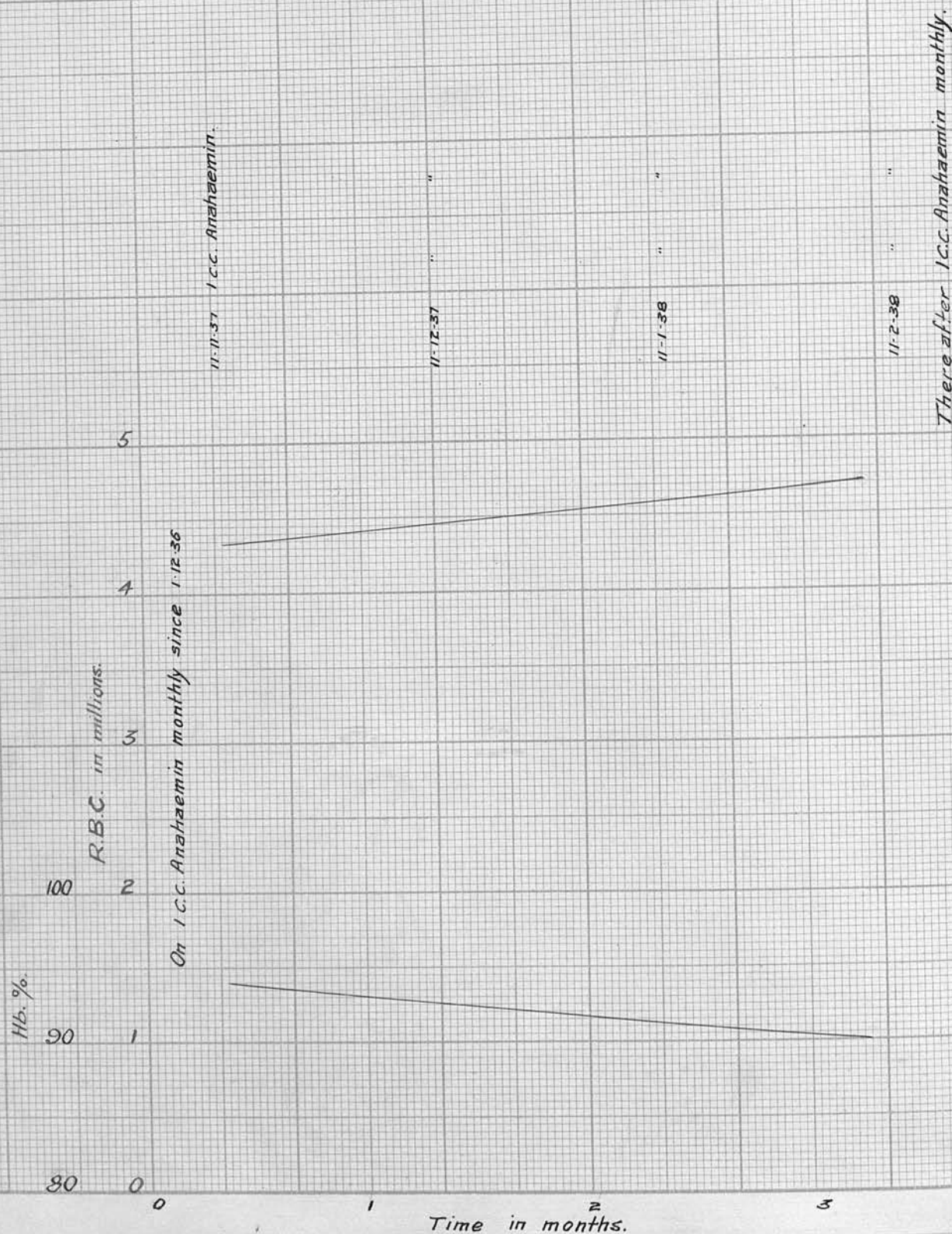
Case 84: was diagnosed by me in October 1936. Her blood pressure had reached a satisfactory level after 4 c.c. of anahaemin were given. She received a maintenance dose of 1 c.c. anahaemin approximately every 4 weeks or slightly oftener. After one year the blood picture was quite satisfactory.

Case 85: was diagnosed in 1932. Hepatex orally failed to keep up his red cell level. I first saw him in November 1936. After 2 c.c. anahaemin his red cells rose from 2.400.000 to 4.100.000. He received 1c.c. anahaemin at approximately 4 week intervals. This dose was not quite sufficient, and after one year the red cells stood at 3.730.000. He received 4 c.c. Neohepatex. After three weeks the red cell level was 4.770.000. He continued to receive 4 c.c. Neohepatex every three weeks and after three months the red cell level was still fairly satisfactory, namely 4.420.000.

Case 86: This patient had pernicious anaemia which was diagnosed in 1932. She had received 2 c.c. pernaemen forte at 4 weekly intervals until December 1936. From then she had 1 c.c. anahaemin every four weeks. I first saw her in/

Graph shewing progress notes of case N° 86 shewing blood counts and treatment given.

Fig. 13.





in November 1937. The red cell level was 4.330.000. She continued having 1 c.c.

anahaemin every four weeks and three months later (February 1938) the red cell level was 4.710.000.

The hospital cases were,

- (1). A case of pernicious anaemia who had been receiving since July 1936, 2 c.c. campolon approximately every third day until 8.4.37. when the red cell level was 1.980.000. In the next 4 weeks she received 8 c.c. of anahaemin, and the blood picture after one month showed a red cell level of 4.700.000. Thereafter she received 2 c.c. anahaemin every month and after eight months the red cell level was 4.480.000.
- (2). A case who had been observed from May till September 1935 and had received 2 c.c. campolon every second day. The red cells rose from 1.130.000 to 3.600.000 in these 4 months. From July till September 1937 the red cell level rose from 1.330.000 to 3.500.000 and in this time she had had 5 x 4 c.c. anahaemin, i.e. in a period of two months.
- (3). A case who had received 86 injections of 5 c.c. campolon in three months, at the end of which his red cell level was 3.340.000. Thereafter he received 2 c.c. of anahaemin on two occasions and after four weeks he had 4.940.000 R.B.C. per cu. m.m.

Cost of Maintenance of a Case of Pernicious Anaemia.

In the treatment of any long continued disease where substitution therapy has to be adopted for life once the diagnosis has become established, the co-operation between the physician and patient is a matter of absolute necessity.

Pernicious anaemia is such a disease. We cannot cure it, in the strict sense of the word; we can simply continue to exhibit the principles necessary for healthy erythropoiesis.

There is the further complication that the patient's symptoms are, unfortunately, not an infallible guide to the state of his blood picture, and that unless his blood count is done at regular intervals he may relapse to a dangerous degree. This necessitates, therefore, regular and frequent consultations with the family doctor or specialist.

These frequent consultations, with the consequent doctor's bill, form a serious obstacle to adequate treatment among the poorer classes.

Another factor of considerable importance is that the subject of pernicious anaemia is usually of such an age that his earning power is waning considerably.

The ideal form of therapy is, therefore, a highly potent injectible extract which is only necessary at reasonably long intervals.

In working out the average cost of maintenance of the seven cases given below I have not considered the question of the physician's fee (although, as I have indicated/

indicated this is a matter of considerable importance in poorer class practice) but I have simply confined my attention to the price of the liver extract in an attempt to discover the cheapest form of therapy which is of a reasonably high standard of efficacy.

In buying ampoules of liver extract for injection the greater the number of ampoules ordered the less is the average cost per ampoule, e.g. a box of 100 2 c.c. campolon ampoules costs 99/-, which is an average cost of less than 1ld per ampoule; whereas a box containing 5 x 2 c.c. campolon cost 6/6d., which is an average of  $1/3\frac{1}{2}$ d per ampoule. As most of my patients had a very limited capital at their disposal they were forced to buy liver extract in small quantities at frequent intervals, as their pension or other weekly allowance permitted. This was, actually, a more expensive method of buying, but, unfortunately, very few of them could produce a sum anywhere in the region of £5. My figures for average cost will, therefore, be slightly higher than those given by hospitals and other institutions, where the more economical method of buying in bulk is adopted.

- (1). The first case took raw ox liver, which costs approximately 1/4d per pound. He was taking  $\frac{1}{2}$ lb. daily. This cost him approximately/



approximately 4/6d per week. Although I told him that injections would be cheaper he preferred to take raw liver as he said it was "a food as well as a drug" and he rather liked it!

- (2). In this case the blood reached a satisfactory level after 4 c.c. of anahaemin were given. She received 4 c.c. more to establish a depot, and thereafter 1 c.c. monthly.

3 x 2 c.c. anahaemin costs 15/-d.  
3 x 1 c.c. do. do. 9/-d.

Therefore 10/- worth of anahaemin was sufficient to bring her blood figures up to normal. She received a further 10/- worth and thereafter 3/- per month was sufficient to maintain the improvement. From the initial blood examination in October 1936 till that in October 1937 she received

4 x 2 c.c. and 10 x 1 c.c. anahaemin.

At retail price the patient actually bought

4 ampoules at 5/- each = £1.  
10 do. 3/- each = £1.10/-

The maintenance plus the initial treatment for 12 months cost her £2.10/-, which is an average of 4/4d per month - about/



about 1/- per week.

The actual maintenance dose alone cost 3/- per month - little more than a penny a day.

- (3). This patient was advised to take 4 oz of Hepatex per week (which is not a large dose) This costs 12/6d per 4 oz. bottle. (He just failed to keep up his improvement on 1 c.c. anahaemin - 3/- - monthly).

Thereafter he maintained a fairly normal red cell level on 4 c.c. Neo Hepatex every 3 weeks.

3 x 4 c.c. Neo Hepatex costs 6/6, so that one ampoule costs the patient 3/3d. The weekly cost to this patient was 1/1d (slightly more than the cost of 1 c.c. anahaemin per month).

- (4). This patient kept up very well on a monthly dose of 1 c.c. anahaemin, which represents as shown, less than 1/- per week

In working out the figures for the hospital cases I am again assuming that the patient cannot afford a larger initial outlay than 35/-. Buying ampoules by the gross, as hospitals do, entirely alters, of course, the figures quoted.

- (1). This patient received 37 x 2 c.c. campolon in the time interval quoted.

25 x 2 c.c. campolon costs 27/6d.

5 x 2 c.c. do. do. 6/6d.

Assuming she bought 1 box of 25 ampoules and/

and 2 boxes of 5 ampoules, the cost would be 40/6d. This patient received 40/- worth campolon in five months (8/- per month) but throughout that time her blood count fell to 1.980.000.

Within the next month her red cells rose to 4.710.000. and she received 8 x 1 c.c. anahaemin in this period.

1 c.c. anahaemin costs 3/-  
8 x 1 c.c. do. do. 24/-.

Thereafter she received 2 c.c. anahaemin every month (costing 5/-). Her red cell level was still satisfactory after 8 months and the cost of liver extract was only 5/- per month.

- (2). This case had received in 4 months, approximately 60 injections of 2 c.c. campolon. Assuming that she had at her command a sum of 30/- at one time, we can imagine her buying this amount throughout 4 months in 4 lots, as follows:-

2 boxes of 25 ampoules of 2 c.c.	
(costing 27/6 each).....	56/-
2 boxes of 5 ampoules of 2 c.c.	
(costing 6/6 each).....	13/-
Total cost for 60 ampoules.....	69/-

In four months this is an average cost of 16/10d per month, and in this time the

blood count only reached 3.600.000. In

(1). The second period of observation she received/

received 5 x 4 c.c. (= 10 x 2 c.c.)

anahaemin. This costs 50/- and was given in a period of  $2\frac{1}{2}$  months. The average cost per month was therefore 20/-.

In this case the average cost was more but an equally good response was obtained in half the time with anahaemin.

(3). In the third case 86 injections of campolon were given in  $3\frac{1}{2}$  months.

15 x 5 c.c. campolon cost 35/-.

Assuming that the patient has such a capital, the cheapest method of obtaining this large amount of campolon is by purchasing 6 boxes (two per month).

6 such boxes cost £10.10/-, an average cost of £3 per month. The red cell level at the end of that period was 3.340.000. (6.4.37). The last injection of campolon was given on 24.3.37.

On 17.4.37. and on 19.4.37. he received 2 c.c. anahaemin, and the red cells rose to 4.940.000. The anahaemin cost 10/-.

Unfortunately this patient left the hospital and further notes were not obtained.

### Summary.

(1). The results of treatment of 7 cases of pernicious anaemia/

anaemia are noted.

(2). One case of 10 years' duration is described.

The treatment was raw liver orally.

(3). The average cost of treatment in 6 cases receiving intra muscular injection of liver extract.

(4). It is shown that the intra muscular injection of liver extract is cheaper and more efficient than the exhibition of raw liver orally. The former costs about 1/-, and the latter 4/-, per week.

(5). It is suggested that anahaemin is a much more potent and more economical preparation than campolon.

(6). A case is quoted who was treated by anahaemin and observed over a period of one year. The cost of maintenance was 3/- per month.

(7). A case is quoted with a similar maintenance cost of anahaemin observed over a period of three months.

(8). A case is quoted where an average cost of 5/- worth of anahaemin per month ensured a satisfactory blood picture over a period of 8 months.

(9). A case is shown which, when observed over one year, failed quite to maintain a satisfactory blood picture on 3/- worth of anahaemin per month.

(10). This patient, who is a case of pernicious anaemia of 6 years' duration, did however, maintain a satisfactory blood picture over four months at an average cost of 1/1d worth of Neo Hepatex per week.



Anaemia in Males.

Out of all the 88 patients I examined, only seven adult males were anaemic. They were cases 52, 70, 74, 77, 79, 83, and 86.

An idiopathic hypochromic anaemia occurring in an adult male is a rare phenomenon. I had no such case.

5 CASES OF HYPOCHROMIC ANAEMIA IN ADULT MALES.

states (52).

"Anaemia for which no cause can be found is very uncommon in men. In studying hospital statistics of over four hundred cases of anaemia, I have found only two other cases of unexplained secondary anaemia in men."

He then proceeds to give notes on five cases of anaemia in males in whom no other disease was detected.

In my group of seven cases, two had pernicious anaemia, and each of the remaining five had some underlying pathology. Of these, two were associated with peptic ulcer, one with nephritis, one with haemorrhage and possibly an element of scurvy, and one with lead poisoning. I propose now, briefly to discuss these five cases.

Case 70. This patient was a male who suffered from hyperchlorhydria associated possibly with a peptic ulcer. He showed only a mild degree of anaemia (Haemoglobin 85%).

Case 79. This patient showed a moderately severe anaemia (Haemoglobin 73%). He had undergone radical surgical treatment for a gastric/

### Anaemia in Males.

Out of all the 86 patients I examined, only seven adult males were anaemic. They were cases 50, 70, 74, 77, 79, 83, and 85.

An idiopathic hypochromic anaemia occurring in an adult male is a rare phenomenon. I had no such case. Witts, who has reviewed the subject exhaustively states (58 ).

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Case 79. This patient showed a moderately severe anaemia (Haemoglobin 72%). He had undergone radical surgical treatment for a gastric/

gastric ulcer 10 years previously. No form of iron therapy was tolerated.

Cousin and Hurst ( 59 ) reported a case of Non-Addisonian anaemia, associated with achlorhydria, occurring in a male aged 53, fifteen years after a gastrojejunostomy had been performed for a duodenal ulcer.

Taylor et al. ( 60 ) quote an incidence of achlorhydria and anaemia in 44% of a series of cases following gastrectomy.

Case 77. This was a man of 59 who had a moderate degree of anaemia (Haemoglobin 79%) associated with a chronic nephritis. The results of therapy are not indicated but Heath, in an investigation into the effect of iron therapy in hypochromic anaemia quotes four cases in which there was no response to treatment. ( 61 ).

The American workers Parsons and Ekola-Strolberg ( 62 ) have made an elaborate study of "anaemia in azotemia." They refute the idea that the anaemia which is usually present in the advanced state of nephritis is due to simple dilution of the blood corpuscles, and state that an actual reduction in their numbers is present.

They have made a careful study of the blood chemistry in 42 cases of anaemia associated with nephritis, and came to the following conclusion.

"The/

"The close parallel between anaemia and azotemia, however, is very obvious. So much so that haemoglobin values around 50% have the same prognostic significance as creatinine values of 5 m.g. or more."

We do not, however, estimate that haemoglobin level as routine in assessing the prognosis of a particular case of nephritis in this country.

Case 74. This patient was of unusual interest in that he was a case of severe anaemia, apparently not a pernicious anaemia, occurring in a male. That his anaemia was partly, at least, due to haemorrhage was evidenced by the presence of prolapsed ulcerated haemorrhoids of severe degree, and the demonstration of blood in his stools. Although his cells were markedly reduced (2.970.000) his haemoglobin was even more so (40%) and the low colour index (0.68) was typical of that found in a case of chronic haemorrhage. The case was an unsatisfactory one in that I failed to establish a diagnosis.

Purpura haemorrhagica was a possibility.

An enlarged spleen is in favour of this.

In taking his blood pressure, however I did not elicit a crop of purpuric spots and the capillary resistance test was negative. The platelet count was not done. I did not think that the bleeding time was prolonged/



prolonged and the blood clotted in a normal manner.

Acholoric jaundice was unlikely as the icterus cleared up quickly though the blood picture remained unchanged.

The leucocyte count was 6.200, 5.200, 7.200 and 5.800 on four different occasions over a year. There was not enough evidence of aplastic anaemia. Indeed the blood count did improve on occasion and polychromasia was noted. The various leucocyte percentages were not disturbed, and an aleukaemic leukaemia was very improbable.

The leucocyte count ruled out agramulocytic angina and haemophilia was unlikely. No neoplasm was demonstrated and the patient actually gained weight. He refused ancillary aids to diagnosis such as test meals and X-rays which necessitated hospitalisation.

Splenic anaemia is not a permissible diagnosis until every other condition has been eliminated.

Hodgkin's Disease is a possibility, but though the spleen and liver are slightly enlarged no enlarged lymphatic glands were discovered.

In favour of a diagnosis of scurvy were,

1. The/

1. The lack of Vitamin C in the diet.
2. The presence of petechia and ecchymosis.
3. The blood picture.
4. The pain in the joints and calf muscles.

I treated this man, in the absence of more complete knowledge, as a case of severe anaemia, aggravated by if not entirely due to, chronic and repeated haemorrhage. The haemorrhage presumably came from his ulcerated haemorrhoids and was probably aggravated by a lack of Vitamin C.

He responded at first to iron therapy, and his haemoglobin increased by 20% in one month. He improved very slowly and gradually over the next three months, but refused finally to take any more medicinal iron.

I failed to get him to take orange juice so that I had no opportunity to observe its effect. Dunlop and Scarborough (63) demonstrated conclusively the dramatic haemopoieses which results from the simple addition of ascarbic acid to a diet in which this vitamin has been shown to be deficient.

From November 1937 till February 1938 three months' intensive iron therapy was enforced. Ferrous sulphate gr. xviii daily were/

were administered. The haemoglobin rose by only 14% in those three months, and the red cells from 3.040.000 to 3.760.000. Since writing up my clinical notes, two weeks ago, I have been called in urgently to see this patient. He developed a sudden and acute attack of asthma. This was quickly relieved by the injection of  $\frac{1}{2}$  c.c. of adrenaline 1/1000 given subcutaneously. He suffered five such attacks in three days. They were relieved by taking a lobelia and stramonium mixture thrice daily. Ephredrine gr.  $\frac{1}{2}$  orally has aborted an attack on two occasions. He had never had **such** an attack in his life before. After the first three days he remained free from an attack for one week. In the interval I could detect no abnormality in the chest. In view of the above findings, however, I am considering the possibility of an atypical form of Hodgkin's Disease with the mediastinal glands chiefly affected. I could not demonstrate, however, any of the classical signs of enlarged mediastinal glands. years. I can only conclude that The patient had another attack of asthma two days ago, and I took the opportunity while/

while he was ill of persuading him to go to hospital for further investigation.

I cannot understand how he allowed me to carry on, for a whole year, treating him with very little tangible success.

This case does not reflect much credit on my diagnostic acumen but I have included it as it illustrates many of the problems and difficulties encountered by the general practitioner when he is forced, single-handed, to cope with a complicated case of anaemia of unknown origin.

#### Case 50.

was a man of 38 who was an oxyacetylene burner employed in a shipbreaking yard.

I decided that he was suffering from chronic lead poisoning from the following findings-

1. Weakness of extensors of both wrists.
2. Constipation.
3. Colicky pain in the abdomen.
4. Anaemia of moderate degree (Haemo-globin 81)
5. Punctate basophilia in the blood film.
6. The nature of the patient's occupation.

There are many men employed locally as lead burners and ship painters. This is the only case of lead poisoning, and it is of mild degree, which I have seen in the course of two years. I can only conclude that the prophylactic measures adopted at the works are very efficient.

Although many observers regard the anaemia



of lead poisoning as a dyshaemopoietic anaemia due to the toxic effect of lead on the bone marrow, Aub. Resnikoff and Smith ( 64) regard it as a haemolytic anaemia. They state that the chief action of lead is upon the surface of the red blood corpuscle, causing it to be brittle, more fragile and more easily haemolysed.

Brookfield ( 65) studied the blood changes occurring during the course of treatment of malignant disease by lead. He is of the opinion that the stippled cells are immature cells altered by the action of lead.

#### Summary.

1. Anaemia in adult males is uncommon.
2. An unexplained anaemia in adult males is a rarity.
3. 7 cases of anaemia in adult males are recorded.
4. Two of pernicious anaemia have been described elsewhere.
5. Two cases of anaemia associated with peptic ulcer are noted.
6. One case of anaemia associated with nephritis is described.
7. One case of anaemia associated with haemorrhage is described.
8. A case of chronic lead poisoning with anaemia is described.

## Idiopathic Hypochromic Anaemia.

It is proposed to discuss the cases of idiopathic hypochromic anaemia as a whole.

There were 30 cases of hypochromic anaemia in whom no obvious organic disease was detected. The

### 44 CASES OF HYPOCHROMIC ANAEMIA IN ADULT FEMALES.

20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30.

a. Idiopathic hypochromic anaemia -  
30 cases.

b. Anaemia associated with pregnancy.  
6 cases.

Four of these (33, 46, 66, and 67) had already

been treated. c. Anaemia following haemorrhage -  
2 cases.

d. Anaemia associated with focal sepsis.  
4 cases.

e. Anaemia associated with duodenal ulcer.

f. Anaemia following deep X-ray therapy.

described as Simple Anchlorhydric Anaemia, Vitale's

Anaemia, Chronic Microcytic Anaemia, Primary Hypo-

chromic Anaemia, Fabry's Syndrome, Idiopathic Hypo-

chromic Anaemia, Chronic Chlorosis, and Achylia

Gastrica.

In a recent textbook (66) the following definition is given.

"A chronic anaemia chiefly found in middle-aged women, characterized by a low mean corpuscular haemoglobin concentration and mean corpuscular volume resulting in a low colour index; achlorhydria and glossitis are usual, while xerophthalmia is common."

Harris himself (67) gives the following definition.

Idiopathic Hypochromic Anaemia.

It is proposed to discuss the cases of idiopathic hypochromic anaemia as a whole.

There were 30 cases of hypochromic anaemia in whom no obvious organic disease was detected. The case numbers are

20, 21, 23, 27, 28, 32, 33, 37, 39, 40,  
41, 46, 47, 48, 49, 51, 52, 54, 55, 57,  
58, 63, 64, 65, 66, 68, 71, 72, 78, 80.

Four cases (Nos: 23, 48, 65, and 68) had already been taking iron before I examined the blood.

It is proposed therefore, to study only the remaining 26 cases.

The group which we are discussing fits, more or less, into a syndrome which has been variously described as Simple Achlorhydric Anaemia, Witts' Anaemia, Chronic Microcytic Anaemia, Primary Hypochromic Anaemia, Fabers' Syndrome, Idiopathic Hypochromic Anaemia, Chronic Chlorosis, and Achylic Chloranaemia.

In a recent textbook (66) the following definition is given,

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Witts himself (67) gives the following definition./

definition.

"Simple achlorhydric anaemia is a form of anaemia which is not uncommon in women and occasionally occurs in men. It is most frequent in middle and later life. The cardinal feature of the disease is the absence of free hydrochloric acid from the gastric juice. Glossitis occurs in many cases and the spleen may be slightly enlarged. The anaemia is of the microcytic type and the colour index is low."

A syndrome of anaemia dysphagia and splenomegaly was described in America by Plummer and Vinson in 1921.

The condition was first recognised in this country by A.F. Hurst in 1926 (68). He added 'glossitis' to the syndrome. His paper is worthy of quotation - he states,

Under the title of "Hysterical Dysphagia" Plummer and Vinson of the Mayo Clinic described in 1921 a remarkable syndrome which had not hitherto been recognised. Among 69 cases of functional dysphagia, 37 had a "secondary anaemia" with haemoglobin varying between 27 and 60 %, and 12 splenomegaly. So far as I know no other cases of this kind have been reported. The following case, which corresponds very clearly with Vinson's description, is therefore worth recording, though the streptococcal glossitis, which was a very prominent symptom, was not described by Plummer and Vinson."

Hurst mentions the fact that Vinson was of the opinion that the dysphagia was hysterical and that the unbalanced diet caused the enlargement of the spleen and secondary anaemia. From the observation of his own case he was of the opinion that the unbalanced diet was not the cause of the anaemia. The dysphagia he said, was due to reflex spasm caused by the local irritation by the streptococci which caused the glossitis./



glossitis. The anaemia and splenomegaly were, in his opinion, due to the streptococcal infection and general focal sepsis.

### Etiology of Hypochromic Anaemia.

All authorities are agreed that the hypochromic anaemia which has just been defined, is due, partly at least, if not wholly, to an inadequate assimilation of iron.

J. F. Brock (69) in a recent article sums up in the following succinct manner.

"The repeated association of hypochromic anaemia with conditions which would be expected to lead to iron deficiency, and their speedy cure with iron therapy, strongly favours the view that hypochromic anaemia is conditioned by iron deficiency."

The role of deficient assimilation of iron is not denied, but the importance of other dietary deficiencies and of the presence of achlorhydria have been frequently debated at great length. Witts, who is an authority on the subject states (70).

"The achlorhydria is a primary aetiological factor in the disease. It is found before the development of the anaemia and persists when the anaemia is cured."

Davidson and Fullerton (71) in an article published only one month ago, are inclined to minimise the role of achlorhydria as a primary cause of anaemia. They sum up,

"The view that the achlorhydria associated with chronic nutritional hypochromic anaemia is familial in origin and precedes the anaemia is not accepted. Evidence is presented in favour of the/

"the hypothesis that the gastric defect, the anaemia, and the nutritional changes are primarily conditioned by the same cause, namely, long-conditioned dietary deficiency."

In a recent address on achlorhydria Eason (72 ) found that constipation, anaemia, and cholecystitis were the most frequently associated disorders. He states that there is a tendency for a patient with achlorhydria to eat a high carbohydrate diet at the expense of protein and fat. He believes that achlorhydria may be an important factor in the aetiology of anaemia in so far as it effects the peptic digestion and leads to a disproportionate ingestion of starchy foods with a reduction in iron foods and a reduced absorption of iron.

Davidson (op. cit. ) is of the opinion that the matter can be traced further back. He reverses Eason's statement and states that the high carbohydrate diet is primary, and is enforced by poverty. The diet of the poor is rich in carbohydrates and astringents (tea). It is, however, poor in protein, mineral salts including iron compounds, and vitamin. The physical nature of the food and the manner in which it is eaten, often renders it indigestible. Such a deficient diet usually favours focal sepsis in the mouth and nasopharyngeal areas.

During a discussion on high carbohydrate diet J.H.P. Paton quoted figures from an investigation of 450 school children (73 ). He was of the opinion that/

that the catarrhal rate was roughly proportional to the sugar intake.

J. Eason ( '74) is of the opinion that the incidence of enlarged tonsils and adenoids may so frequently co-incide with dietetic faults as to indicate that these disorders are, in a large measure, due to nutritional deficiency.

The type of diet then, which is described in detail in many of the cases quoted above leads to degenerative nutritional changes in the gastric mucosa. The diet itself also favours focal sepsis in the upper respiratory tract. Pus is swallowed and damages the mucous membrane of the stomach, whose powers of resistance are already lowered by dietary deficiency. The crowning insult is the arrival of the deficient diet, the physical and chemical nature of which renders it highly indigestible. The net result is a chronic gastritis leading to a hypochlorhydria. This itself leads to interference with absorption of foodstuffs. As an escape from the dyspepsia which ensues a high carbohydrate diet is taken, and a vicious circle is begun.

From my own observation I am inclined to support the view of Davidson, that the deficient diet is the most striking feature which is common to most cases of 'primary' hypochromic anaemia met with in general practice.

Deficient diet alone is not, however, sufficient to/



Fig. 15.

Graph shewing % of cases of severe Anemia (ie. 70% or less Hb.)  
arranged in age groups. 26 cases.

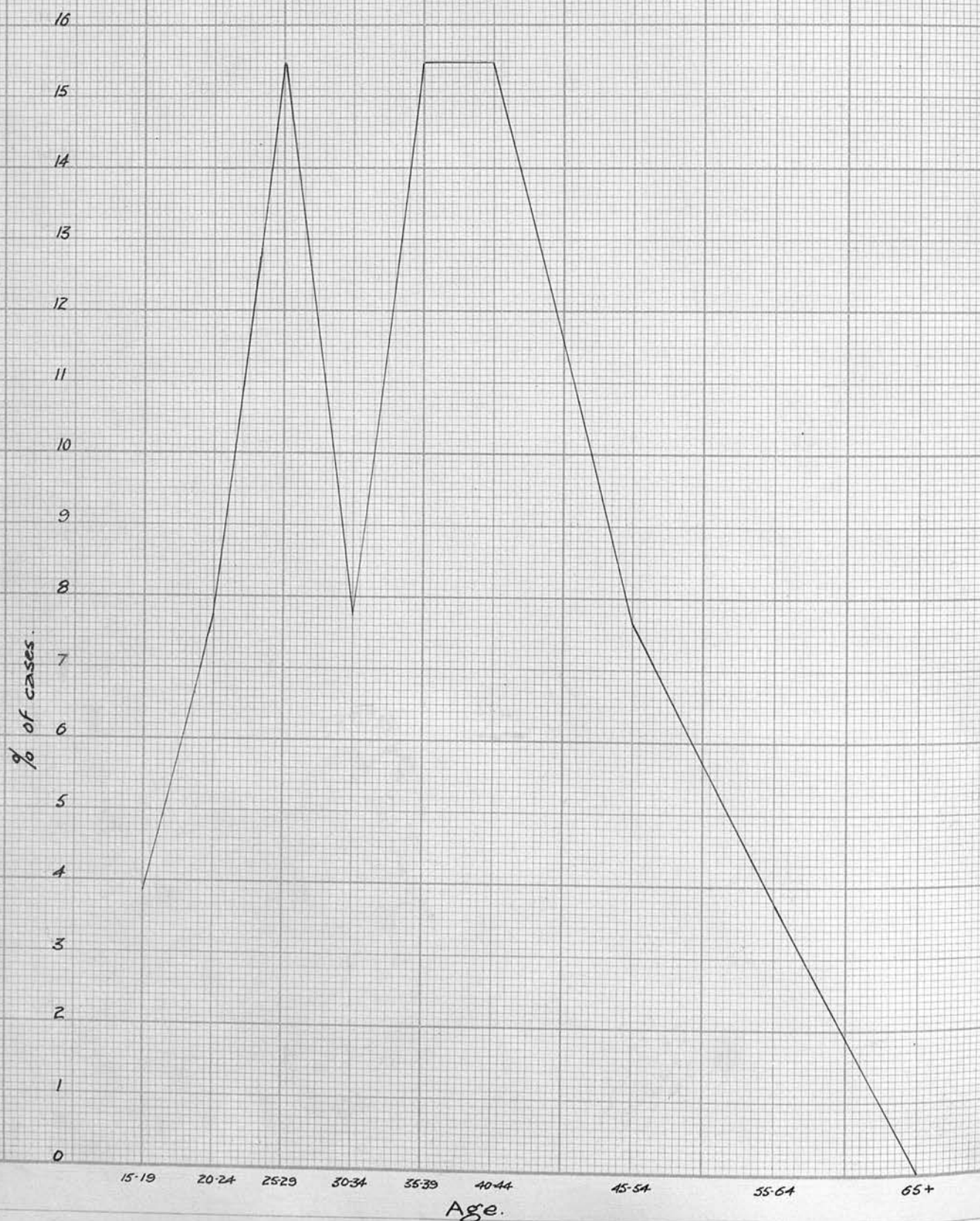
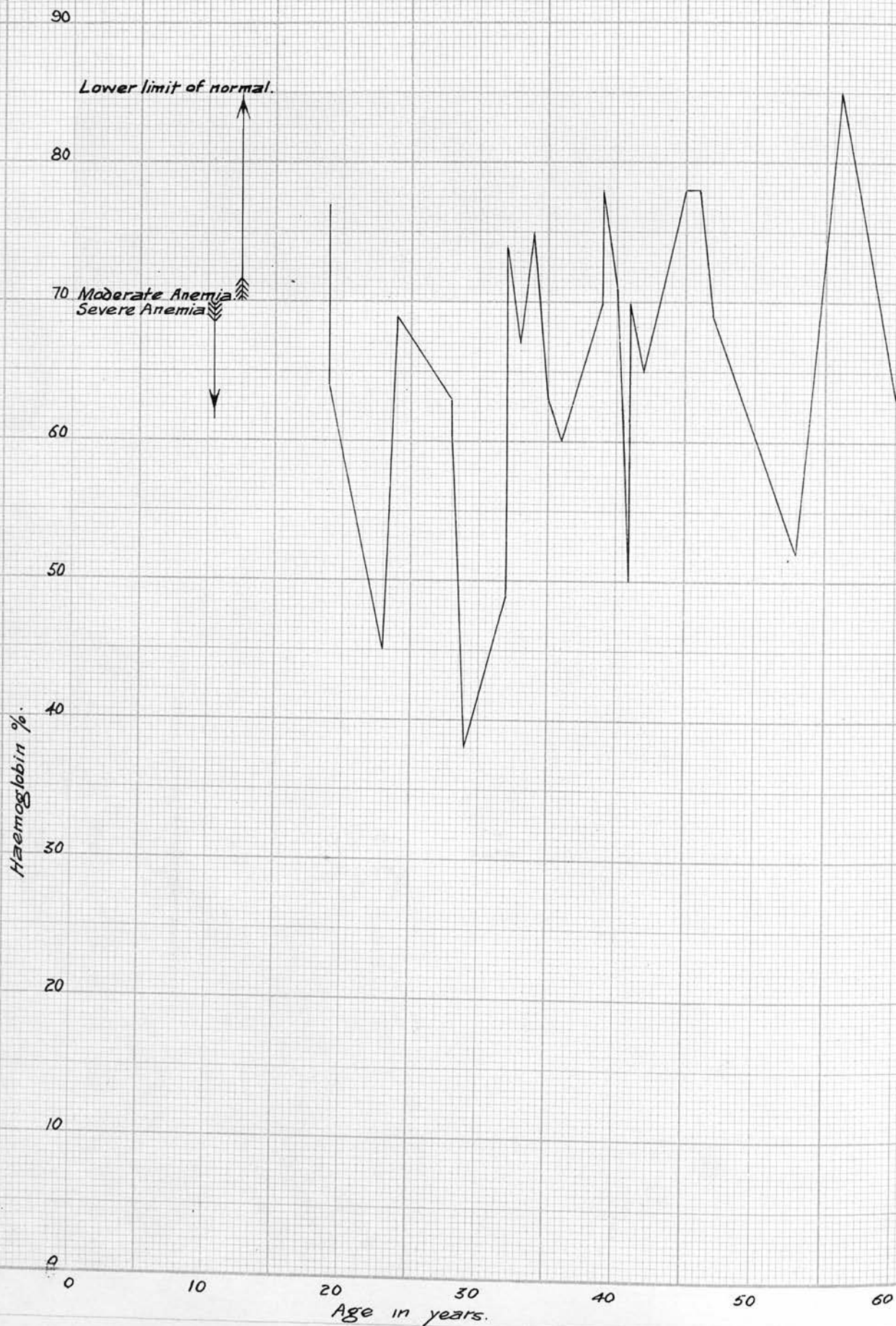




Fig. 14.

Graph shewing relationship between age and Hb. level in 26 cases of Idiopathic Anemia.



to account for the prevalence of anaemia. This is at once obvious when the rarity of the condition among adult males, who are consuming a similar diet, is noted. In my series of 68 cases, only 7 were adult males; of these, two had pernicious anaemia and the remaining five definite demonstrable organic disease. Not one showed an idiopathic hypochromic anaemia.

Some additional factor must be present in females which is absent in males. A survey of my 26 patients shows that the average age of incidence was 37.8 years. A graph is shown (Fig. 14) showing the haemoglobin level obtained in each case with the corresponding age of the patient noted. A more striking fact is revealed in a study of the cases who were severely anaemic, i.e. with haemoglobin percentages of 70 or less. 17 of the 26 cases fell into this class. A graph was prepared showing the percentage (out of 26 cases) of severe anaemia in relation to the age of the patients (Fig. 15). It is observed that there was an incidence of 15.5% in the 25-29 age group; that the incidence was not so high in the 30-34 age group; but the maximum incidence was sustained throughout the 35-44 age group, and thereafter the number of cases of severe anaemia fell away abruptly. From this we can state that the severer forms of this anaemia are confined to the reproductive period of adult life, and that the highest incidence occurs in and around the age/

age of 40. After the menopause the incidence of severe anaemia falls away rapidly. These figures are based on the findings of 26 cases, but since compiling them I have read in a very recent publication by Davidson and Fullerton that in a review of 715 parous, non-pregnant women whose ages ranged from 15 to 65 years, they found that (75 ).

"the haemoglobin level falls gradually with increasing age up to the menopause, and then rises markedly. The incidence of anaemia in women of the reproductive age was very high. 48 per cent showed readings less than 85 per cent, and 16 per cent were severely anaemic (haemoglobin less than 70 per cent). After the menopause the incidence of anaemia was much less."

This statement lead Davidson and his colleagues to believe that

"the frequency of anaemia in adult women is due mainly to the demands of reproductive life, namely menstruation and pregnancy."

Fullerton has shown (76 ) that the average daily intake of iron in women of the poorer classes in Aberdeen is 8.57 Mg. He allows 10% for wastage in cooking which gives him a figure of 7.71 Mg. Making further correction for an erroneous calculation of the iron in "golden syrup" reduces the figure to 7.3 Mg. As only 75% of the iron in foodstuffs is "available" the figure is further reduced to 5 Mg. and, he states, about half of this only will be absorbed. Hence he states 2.5 Mg. is a fair average of the amount of iron absorbed daily from the diets of his patients. 2.5 Mg. is /



is equivalent to an iron assimilation of 70 Mg. in 28 days.

A menstrual loss of over 70 Mg. occurring every 4 weeks will, therefore, create a negative iron balance. Fullerton quotes the figures of many American investigators of the average blood loss of menstruation.

(77) Fowler and Barer found a wide range of iron loss at menstruation in women who considered that their loss was normal. It extended from 3.5 to 450 Mg. In a series of 10 anaemic women the average loss was in the region of 120 Mg. The iron loss is, therefore, extremely variable, but it appears that anaemia will result in a patient consuming the type of diet which Fullerton found to be common in Aberdeen, if the iron loss is greater than 70 Mg. at each menses.

With regard to the other possible source of iron loss in women, namely pregnancy, parturition, and the puerperium, Fullerton in a later article states (78) that

"The conception that uncomplicated pregnancy frequently produces a severe degree of hypochromic anaemia should be discarded."

He adds however, that

"the blood loss at parturition varies greatly in degree and often produces severe hypochromic anaemia."

He states that an average iron loss during pregnancy and the six months after delivery is 905 Mg. apportioned as follows,

Uterus and contents.....	550.
Blood loss at parturition.....	175.
Six months' lactation.....	180.

Total.....905 Mg.//



Total.....905 Mg.

This loss occurs over a period of 460 days, and represents an average daily loss of just under 2 Mg. which even a poor class diet can cope with.

Further he points out, that 460 days correspond to 16 menstrual periods, and shows that 16 menstrual periods with an average loss of 57 Mg. is equal to the iron loss of pregnancy and the puerperium.

As a logical conclusion to this he states that a positive balance of 2 Mg daily iron, will prevent the development of anaemia due to pregnancy.

As many of his cases failed to achieve such a balance he discovered that repeated pregnancies did produce an iron deficiency anaemia.

He found, however, that in many cases the iron loss by menstruation actually exceeded that caused by pregnancy and its sequels.

He was, therefore, able to show that there was no relation between parity and the degree of anaemia present.

The conception that severe anaemia was more common in multipara than nulliparous patients was simply due to the fact that severe anaemia was more common in the early forties than in the early thirties. The older the patient, on the whole, the more children she had borne.

If cases were compared who belonged to the same  
age /

age groups it was found that no relation could be established between the parity and the degree of anaemia present.

In connection with pregnancy there is one final point I should like to make. That is the findings of Strauss and Castle (79) that the secretion of hydrochloric acid is markedly reduced during pregnancy and as it is well known that the absorption of iron is enhanced by the presence of hydrochloric acid, we can assume that a decreased absorption of an already too insufficient dietary iron will frequently result during pregnancy, and be increased by repeated pregnancies.

#### The Symptoms and Signs in 26 Cases of Idiopathic Hypochromic Anaemia.

At this point I shall give (for the readers' further reference) the case numbers, age, haemoglobin level, red cell percentages and colour index of my 26 cases.

They are/

The incidence of cases increased from childhood

They are

<u>Case Nos:</u>	<u>Age.</u>	<u>Haemoglobin.</u>	<u>R.B.C.</u> <u>(in millions)</u>	<u>Colour</u> <u>Index.</u>
80.	62.	75.	5.24.	.72.
78.	60.	63.	4.69.	.68.
72.	56.	85.	4.59.	.94.
71.	53.	52.	4.26.	.61.
66.	47.	69.	4.93.	.70.
64.	46.	78.	4.41.	.89.
63.	45.	78.	4.68.	.85.
58.	42.	65.	3.85.	.84.
57.	41.	70.	4.62.	.76.
55.	41.	50.	3.33.	.76.
54.	40.	71.	4.29.	.85.
52.	39.	78.	4.64.	.85.
51.	39.	70.	4.53.	.78.
49.	36.	60.	3.94.	.76.
47.	35.	63.	4.1.	.77.
46.	34.	75.	4.4.	.85.
41.	33.	67.	4.25.	.81.
40.	32.	74.	4.91.	.76.
39.	32.	49.	4.01.	.61.
37.	29.	38.	2.82.	.68.
33.	28.	60.	3.8.	.79.
32.	28.	63.	4.02.	.79.
28.	24.	69.	4.24.	.82.
27.	23.	45.	4.05.	.56.
21.	19.	64.	3.7.	.87.
20.	19.	77.	5.03.	.77.

Average - 37.80. 65.7. 4.1923. .77, ~~1923~~

Sex: All cases were females.

Age: The ages varied from 19 to 62 and the average age was 37.8 years. The arrangement of cases according to age groups reveals the following,

Under 20	years,	show	no: of cases.....	2
20 - 29	do.		.....	5
30 - 39	do.		.....	8
40 - 49	do.		.....	7
50 - 59	do.		.....	2
60 - 69	do.		.....	2

Parity: The incidence of cases increased from adolescence up/

up to the age of 30. There was a high incidence in the 30 - 39 age group and almost as high a figure in the 40 - 49 group. After 50 years the incidence fell away rapidly.

Of the 26 cases 23 occurred during the active reproductive period of life. Only three cases occurred after the menopause (cases 72, 78, and 80). Of these three, only one was severely anaemic, case 78 (Haemoglobin 63). These findings are in agreement with those of Witts (30) who, in a series of 50 cases found that 8 occurred in the 20 - 29 age group, 14 in the 30 - 39, 21 from 40 - 49, 5 from 50 - 59, and 2 from 60 - 69.

This peak of incidence is in a higher age group, 40 - 49, than in my cases.

If I exclude the milder degrees of anaemia, and take those of my cases showing readings of 70% or less, a graph of the percentage incidence of these cases (Fig. 15) shows that severe anaemia was much more common in the 35 - 44 age group, and that the percentage of cases in this group represented 51% of the total cases



Parity: 9 of my cases were nulliparous; the remaining 17 had borne children. For reference I shall give the case numbers and parity in the 26 cases.

No: of Children.

	0 ;	1 ;	2 ;	3 ;	4 ;	5 ;	6 ;	7 ;
80.	66.	72.	64.	78.	0.	0.	71.	
58.	63.	47.	54.	57.				
52.	55.	27.	49.					
39.	51.							
33.	46.							
32.	41.							
28.	37.							
21.								
20.								
	9.	7.	3.	3.	2.	0.	0.	1.

The age and haemoglobin level of these cases are indicated on page 229.

From these data it was possible to prepare a table showing the relationship between parity and the degree of anaemia present. The haemoglobin levels of women with three children or more were compared with those of women of the same age group who were nulliparous, or had borne not more than two children. Six patients fell into the former class and 20 into the latter.

It was only possible in three age periods - (35 - 44) (45 - 54) (55 - 64) - to obtain full statistics. The results obtained are as noted:-

26 cases belonged to the poorer classes. I have not made a detailed study of the family income

are noted:-

Age Group.	Below 25.		25-34		35-44		45-54		55-64	
Previous Pregnancies.	0-2	3 +	0-2	3 +	0-2	3 +	0-2	3 +	0-2	3 +
No. of Cases.	4	0	7	0	5	3	2	2	2	1
Average Haemoglobin.	63.7	-	60.9	-	65.2	67	78	60	80	63
No. of cases with Haemoglobin less than 70 %,	3	-	5	-	3	1	1	1	0	1

A glance at the table shows that no relationship could be established between the parity of the patient and the degree of anaemia present.

While I have not at my command the number of cases quoted by Fullerton in 1937 (vide supra) my results definitely conform to his findings. I had begun doing blood counts before I read this article which was only recently published, and I was not, therefore, influenced by it in the selection of my cases. Indeed the reasons for selecting these cases are clearly indicated in the case notes above.

#### Social Status.

Fullerton (81) has made an elaborate study of the relationship between the average weekly income and the degree of the anaemia present. He shows that there is a close parallel. Most of my 26 cases belonged to the poorer classes. I have not made a detailed study of the family incomes but/

but have indicated them in a general manner in the case notes. Six of my cases were definitely more financially favoured than the average for the series. Case 28 was a nurse. The other five cases, 40; 46; 51; 54; and 72, were the wives of a fishmonger, an estate factor, a doctor, a dentist, and a contractor, respectively. Their haemoglobin levels were 69; 74; 75; 70; 71; and 85, respectively. The average haemoglobin level for the eight cases was 74% which was considerably above the average for the whole series, viz. 65.7 %.

#### Blood Picture.

The average for the 26 cases was;-

Haemoglobin	65.
R.B.C.	4.192.000.
C.I.	0.77.

#### Dyspnoea, Palpitation, and Oedema.

These were present only in the severer cases and showed a definite relationship to the degree of anaemia present.

#### Pallor

of the mucous membranes was present in every case but there was often no correlation, especially in the milder cases, between the pallor and the degree of anaemia present.

#### Dysphagia

was present in 9 of the 26 cases and was a prominent/

prominent symptom in four cases, (Nos; 27, 37, 46, and 55), giving a percentage incidence of 15%. The haemoglobin levels of these cases were 45, 38, 75, and 50 respectively, with an average haemoglobin level of 52% which is considerably below the average for the series.

In a series of 125 cases of severe anaemia Davidson and Fullerton (82) found that dysphagia was a prominent symptom in 10% of their patients. Witts (80) in a review of 50 cases found dysphagia to be present in 12%.

#### Nail Changes.

Nail changes were absent in 17 cases. Of the 9 cases showing nail changes, 4 cases had definite concave, spoon-shaped nails (Koilonychia). Their case numbers are 37, 49, 55, and 57. The corresponding haemoglobin levels were 38, 60, 50, and 70 respectively, with an average of 54.5 - well below the average for the series. The percentage incidence of koilonychia was 15%. The other five cases showed the presence of cracking and longitudinal ridges. Five of the 9 cases complained of the brittleness of/



of the finger nails.

Witts gives an incidence of koilonychia of 8% and Davidson and Fullerton in their group found that nail changes were absent in 44%, were slight in 39%, and were marked in 17%.

#### Tongue Changes.

Glossitis was present in 7 cases, numbers 40, 41, 46, 55, 58, 63, 64. In four of these the tongue was red and hypertrophied; in the remaining three it was atrophic. The total incidence of tongue changes was 26.9%.

The haemoglobin levels for the seven cases were 74, 67, 75, 50, 65, 78, and 78, giving an average of 69.6%. This was above the average for the whole series, and there was apparently no relation between the degree of anaemia and the incidence of tongue changes.

Davidson found that papillary atrophy was absent in 35% of his cases, was slight in 35% and marked in 30%.

Witts found that glossitis was present in 25 of his 50 cases (50%). He includes all tongue changes under this heading and is of the opinion that the incidence of these changes/

changes is even higher, as they are easily missed if not looked for.

Amenorrhoea. was noted in 5 cases, nos: 27, 37, 39, 41 and 71. The respective haemoglobin levels were 45, 38, 49, 67, and 52% giving an average of 50%, which is well below the average for the series.

5 cases of amenorrhoea gives a percentage incidence of 19%.

Witts had an incidence of 10% in his cases.

Menorrhagia: was noted in three cases, namely numbers 33, 47, and 66. The respective haemoglobin levels were 60, 63, and 69, which gives an average of 64% which is not appreciably different from the average haemoglobin percentage for the whole series ( 65%).

3 cases represent an incidence of 11.5 % as opposed to Witts' figure of 14%.

Dyspepsia: which does not include mere loss of appetite alone, was present in 14 cases, i.e. in 53.8% of the series of cases under discussion. In the series of 50 cases which are described by Witts, achlorhydria was demonstrated in 44 and extreme hypochlorhydria in 6. In this series of cases dyspepsia was a symptom in 15 of them - i.e. in 30%. My case numbers with the haemoglobin percentages/

percentages noted were:- in this case of

<u>Case Nos:</u>	<u>Haemoglobin.</u>
21.	64.
27.	45.
32.	63.
33.	60.
37.	38.
39.	49.
46.	75.
47.	63.
55.	50.
63.	78.
66.	69.
72.	85.
78.	63.
80.	75.

Average.....62.6 %

The average of 62.6% Haemoglobin is not appreciably different from that of the whole series. (65 %).

### Treatment.

Of the 26 cases, seven were treated with Ferri. et. ammon cit.; eighteen with Ferrous sulphate; and the remaining one received both forms of therapy.

#### Ferrous sulphate.

All 19 cases who received Ferrous sulphate gr. ix daily, improved. Follow-up blood counts were only done in 13 of these cases. (nos: 28, 33, 39, 40, 41, 51, 52, 54, 55, 63, 66, 71, and 78). A response of an increase in haemoglobin in the region of 1% per day was obtained in 8 cases; the remaining/

remaining 5 did not attain this rate of haemopoiesis. It is extremely difficult, however, to be sure that the patient is taking the drug exhibited, and this point is borne out fully in the case notes above.

Ferri. et. ammon. cit.

Eight cases were treated with Ferri. et. ammon. cit., and all improved (with the exception of case 33) in a satisfactory manner. Follow-up blood counts were obtained in only 3 cases, and all 3 showed an increase in haemoglobin in the region of 1% daily. One received xxx grains thrice daily (case 64); one received xxv grains (case 58); and one received xx grains (case 21).

#### Summary.

- (1). Idiopathic hypochromic anaemia is defined.
- (2). An original description of the Plummer Vinson syndrome is quoted.
- (3). The aetiology of hypochromic anaemia is discussed.
  - a. The importance of dietary deficiency is stressed.
  - b. The high incidence among females of the reproductive period is illustrated.
  - c. The highest incidence of severe anaemia was found in the 35 - 44 age group.
  - d. The importance of the iron loss of menstruation and pregnancy is stressed.
  - e. The/



- e. The role of hypochlorhydria occurring during pregnancy is mentioned.

(4). The symptoms and signs in 26 cases of idiopathic anaemia are described.

- a. The age incidence is mentioned in detail.
- b. The relationship between parity and the degree of anaemia present is described.
- c. The financial factor is stressed.
- d. The average blood findings for the 26 cases are noted.
- e. Dysphagia was a prominent symptom in 4 cases.
- f. Koilonychia was present in 4 of the cases, and it is shown that anaemia was of greater severity in those cases.
- g. Glossitis was present in 7 cases, and the average haemoglobin of this group was actually above the average percentage for the whole series.
- h. Amenorrhoea was present in 5 cases and the haemoglobin level of this group was well below the average of the series.
- i. Menorrhagia was noted in 3 cases, but the haemoglobin level in this group did not differ from the average haemoglobin percentage for the whole series.
- j. Dyspepsia was present in 14 cases and the average haemoglobin level in this group did not depart appreciably from the average for the whole series.

(5). The treatment of 26 cases is noted.

Anaemia associated with Pregnancy.

6 cases of anaemia associated with pregnancy were noted.

Case 31: was a young mother who had two children  
She had a subacute rheumatism, marked oral  
sepsis, and a mitral stenosis.

The anaemia was severe,

Haemoglobin	56.
R.B.C.	4.860.000.
C.I.	0.58.

and she presented a syndrome of glossitis,  
dysphagia, koilonychia, and dyspepsia very  
early on in the pregnancy. The blood pic-  
:ture taken at this early date, in the  
pregnancy, together with the evidence of  
other organic disease, strongly suggested  
that the pregnancy did not in any way  
condition the anaemia discovered. She  
improved on Ferrous sulphate gr. iii, t.i.d.

Case 45: was a woman of 33 who was five months  
pregnant. The blood picture was,

Haemoglobin.	67.
R.B.C.	4.880.000.
C.I.	0.65.

She also complained of glossitis, dysphagia,  
dyspepsia and brittle finger nails. The  
anaemia preceded the pregnancy but was  
aggravated by it. She improved considerably  
with the exhibition of Ferrous sulphate.

Case 34:/

Case 34: had been delivered only three weeks before the blood was examined. She had only a mild anaemia,

Haemoglobin.	77.
R.B.C.	4.880.000.
C.I.	0.80.

inspite of the fact that her diet was very deficient.

Case 56: was severely anaemic.

Haemoglobin.	45.
R.B.C.	4.170.000.
C.I.	0.54.

She had been delivered 7 months previously.

There was no undue loss at parturition and the anaemia was presumably idiopathic in nature. She failed to improve with the exhibition of Liq. Ferri. Perchlor M xv, t.i.d. She failed to report. One year later the blood figures were not improved, (Haemoglobin 67) ., but she later improved with Ferrous sulphate gr. iii, t.i.d.

Case 29: had been delivered 10 months previously. I examined her blood as a control. She was only a borderline case of anaemia (Haemoglobin 84).

Case 30: was one of severe anaemia following post partum haemorrhage. She made satisfactory improvement with Ferrous sulphate gr. <sup>xviii</sup> ~~xii~~ daily. The blood count 10 months/

10 months after delivery showed the following,

Haemoglobin.	65.
R.B.C.	4.250.000.
C.I.	0.77.

and steadily improved thereafter. One month later the Haemoglobin level was 88 and after three months the figures were

Haemoglobin.	91.
R.B.C.	5.190.000.
C.I.	0.89.

#### Summary.

1. 6 cases of anaemia were associated with pregnancy.
2. Two occurred during pregnancy and 4 were examined after delivery, one at three weeks, one at 7 months and two at ten months.
3. In one case the anaemia was probably entirely due to the pregnancy or rather, a complication of pregnancy, (post partum haemorrhage).
4. In two cases the anaemia had definitely preceded the pregnancy.
5. In the three remaining cases there was insufficient evidence that the anaemia had been caused by the pregnancy.
6. Two of these cases had only a mild degree of anaemia.



Anaemia Following Haemorrhage.

Two cases were observed (nos: 60 and 82).

Case 60: was a woman of 42, the mother of seven children. There was a definite dietary deficiency. Koilonychia was marked. Dysphagia, dyspepsia and menorrhagia were noted. The blood picture was,

Haemoglobin.	42.
R.B.C.	3.920.000.
C.I.	0.54.

She improved rapidly with Ferrous sulphate gr. iii, t.i.d. but had relapsed in one year's time.

Case 82: was an old lady of 82 years. The blood picture was,

Haemoglobin.	38.
R.B.C.	3.490.000.
C.I.	0.48.

Summary.

1. In both cases severe blood loss was taking place from ulcerated haemorrhoids.
2. Both showed a very low colour index.
3. Both improved at once with the exhibition of Ferrous sulphate gr. iii, t.i.d.
4. Dietary deficiency was present in both cases, and in one multiparity and menorrhagia were additional factors upsetting the balance of metabolism.

then Anahaemin parenterally. Her glossitis improved but the anaemia was comparatively unaffected by therapy.

#### Summary.

1. 4 cases of anaemia associated with sepsis are noted.
2. One was a borderline case and was associated with chronic cervicitis.
3. The remaining three were associated with a 'rheumatic' type of focus.
  - a. Tonsillitis.
  - b. Endocarditis.
  - c. Rheumatoid arthritis.
4. In the latter was shown a glossitis of severe degree.
5. Collins ( 83 ) in a review of 100 patients suffering from anaemia associated with 'Rheumatism' found glossitis in 2 cases.
6. In two cases the response to treatment was not known.

Of the other two, one responded well to Ferri. et. ammon cit. and the other failed to respond satisfactorily to either iron or liver therapy.

Campolon & co. at weekly intervals, but no further blood figures are available.

While it is widely recognised that deep X-ray sickness is a clinical entity of considerable importance and that it is often relieved in a miraculous manner by the exhibition of liver/

### Anaemia Associated with Duodenal Ulcer.

This case was a nulliparous woman aged 35 (case 44). She had a **duodenal** ulcer which had been demonstrated radiologically. The Benzidine test was positive. The blood picture was

Haemoglobin.	63.
R.B.C.	4.090.000.
C.I.	0.78.

She did not respond well to treatment. She did not tolerate scale iron. Saccharated Ferrous carbonate was incorporated in her alkali powder but 83 was the highest level to which the haemoglobin level could be pushed.

### Anaemia Following Deep X-Ray Therapy.

Case 67: This was a case of very mild anaemia, bordering on normal figures, which followed the exhibition of deep X-ray therapy. The blood count revealed the following:-

Haemoglobin.	85.
R.B.C.	4.020.000.
C.I.	1.06.
W.B.C.	8.500.

She responded fairly well to the exhibition of Campolon 4 c.c. at weekly intervals, but no further blood figures are available.

While it is widely recognised that deep X-ray sickness is a clinical entity of considerable importance and that it is often relieved in a miraculous manner by the exhibition of liver/

liver extract parenterally there is some doubt as to whether deep X-ray therapy does produce an anaemia.

Radium emanations are very prone to produce anaemia but X-rays are less likely. Thus Rolleston (84 ) reviewed the literature on the subject and found that out of 12 collected cases of aplastic anaemia

7 occurred in radium workers.

1 in thorium workers.

and 4 in those working with X-rays.

Phillips states ( 85 )

"In no cases following massive doses of radiation was there an anaemia produced.

No characteristic changes are produced in the blood by large doses of radiation."



## SUMMARY AND CONCLUSIONS.

1. The observations on which this thesis is based were made by me in the course of routine duties as an assistant in general practice.
2. A description of the type of practice is given and of SUMMARY AND CONCLUSIONS. cases for examination.
3. The desirability of performing blood counts as a routine diagnostic measure in general practice is urged.
4. The normal figures for haemoglobin and red blood corpuscles are stated.
5. The various methods by which the percentage of circulating haemoglobin in healthy blood is calculated, are described in some detail.
6. The Hahnemann scale is defined.
7. The necessity for standardisation of all haemoglobinometers in terms of absolute values is stressed.
8. The reasons for rejecting the uncorrected figures given by my Sahli haemoglobinometer are stated.
9. The standardisation of my haemoglobinometer is described.
10. The phenomenon of the increasing depth of colour in a newly prepared solution of acid haematin is described.
11. Figures obtained from an investigation of this phenomenon/

SUMMARY AND CONCLUSIONS.

1. The observations on which this thesis is based were made by me in the course of routine duties as an assistant in general practice.
2. A description of the type of practice is given and of the method of selection of cases for examination.
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9. The standardisation of my Haemoglobinometer is described.
10. The phenomenon of the increasing depth of colour in a newly prepared solution of acid haematin is described.
11. Figures obtained from an investigation of this phenomenon/

phenomenon are used to establish a method for the quick conversion of readings on the Sahli Haemoglobinometer into percentages on the Haldane scale.

12. The principle of the diffraction method of measuring the mean red cell diameter is described and its limitations outlined.
13. My experience with an instrument of this type are given.
14. The case notes of 86 patients observed over a period of  $1\frac{1}{2}$  years in general practice, are given, together with the blood picture, treatment, and progress notes.
15. Three cases of pernicious anaemia observed in hospital are added.
16. The clinical classification of 68 cases of anaemia in general practice is given. The cases fall into the following groups, according to age.

1. Children.....14 cases.
2. Adolescents..... 1 case.
3. Adult males..... 7 cases.
4. Adult females.....46 cases.

The clinical subdivision of these groups reveal the following:-

- |                                      |           |
|--------------------------------------|-----------|
| Hypochromic anaemia in children..... | 14 cases. |
| do. do. adolescents.,                | 1 case.   |
| Pernicious anaemia.....              | 4 cases.  |
| Hypochromic anaemia in adult males.. | 5 cases.  |
| do. do. females..                    | 44 cases. |

Total....68 cases.

17. When cases of pernicious anaemia and cases with  
a/

a demonstrable organic basis are excluded, only 33 cases of idiopathic hypochromic anaemia remain. Of these 30 occurred in adult females, and 3 in children.

18. It is therefore concluded that an idiopathic hypochromic anaemia in which no underlying pathology is found is common only in children and females.
19. 26 of the cases of idiopathic anaemia in adult females are discussed as a whole.
20. The importance of dietary deficiency and iron loss due to menstruation are stressed.
21. The high incidence of the condition in the re-productive period, and especially towards the menopause is stressed.
22. The role of repeated pregnancies in the aetiology of hypochromic anaemia is minimised.
23. The symptomatology is discussed and the incidence of glossitis, dysphagia, koilonychia, dyspepsia, amenorrhoea, and menorrhagia are noted.
  1. In relation to the total number of cases. (26)
  2. In relation to the degree of anaemia present.
24. The response to treatment is indicated.
25. The other hypochromic anaemias found in adult females are very briefly noted under appropriate headings/



headings.

26. Seven cases of pernicious anaemia are discussed.
27. Two cases of hyperpoiesis associated with pernicious anaemia are described.
28. The financial aspect of pernicious anaemia is stressed, and the average weekly cost of maintenance of the seven cases are noted in detail.
29. It is shown that parenteral injection is a cheaper method of therapy than the exhibition of raw liver orally.
30. It is suggested that Anahaemin is a cheaper and more efficient liver extract than Campolon.
31. Five cases of hypochromic anaemia occurring in adult males are briefly noted under appropriate headings.
32. Fourteen cases of hypochromic anaemia in children are discussed as a whole. The associated pathological lesions are noted.
33. The importance of thorough examination of anaemic children in order to discover the underlying pathology, is illustrated.

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